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SUTUREGRAM

NUMBER SIXTEEN (CONTINUED)

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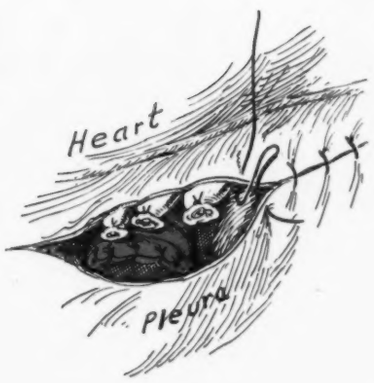

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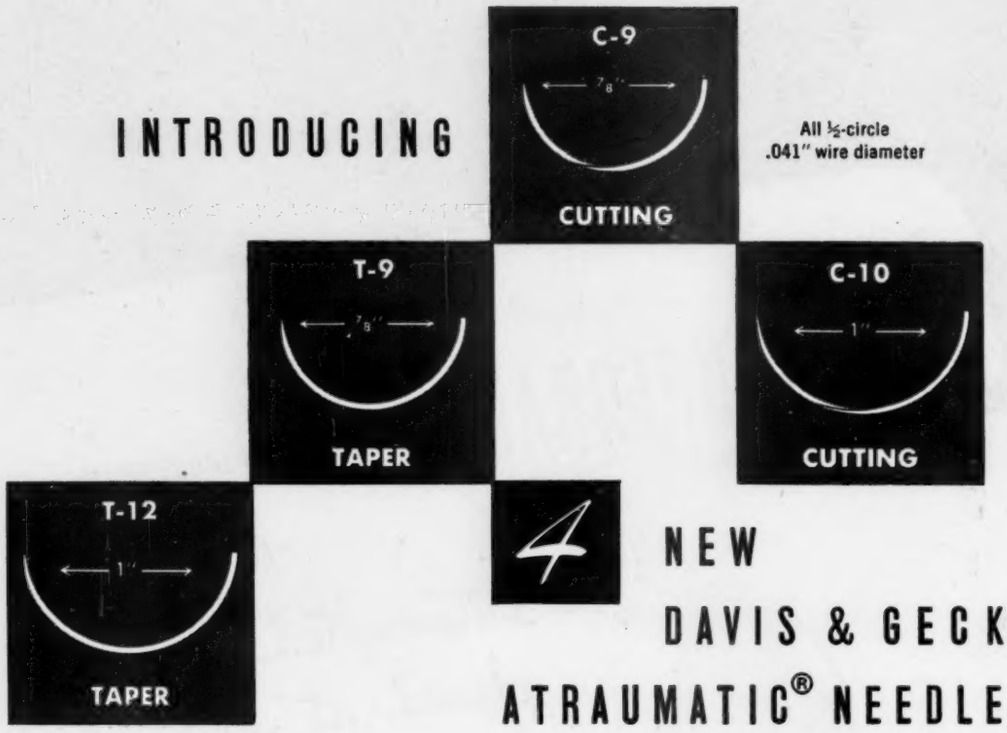

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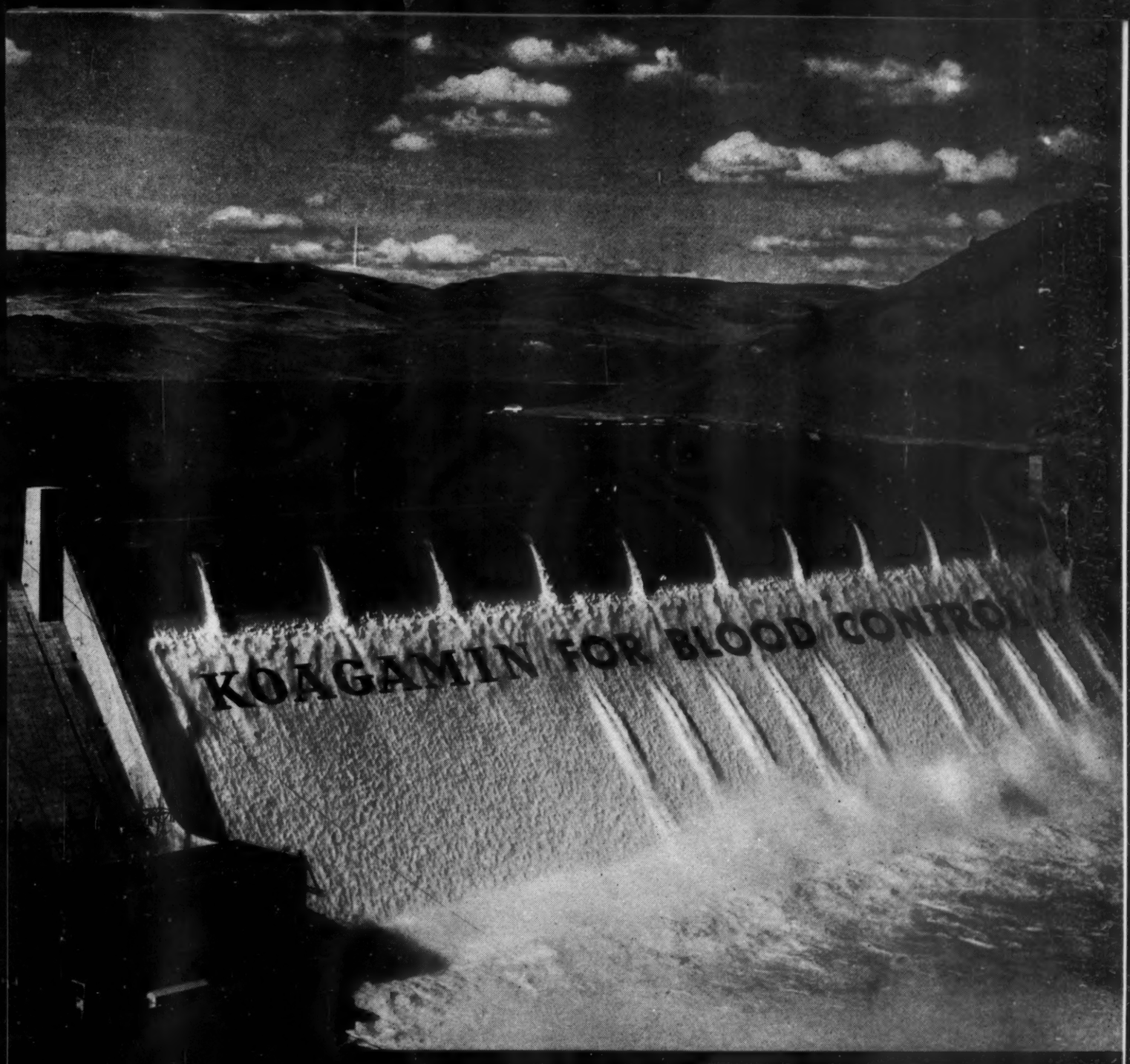

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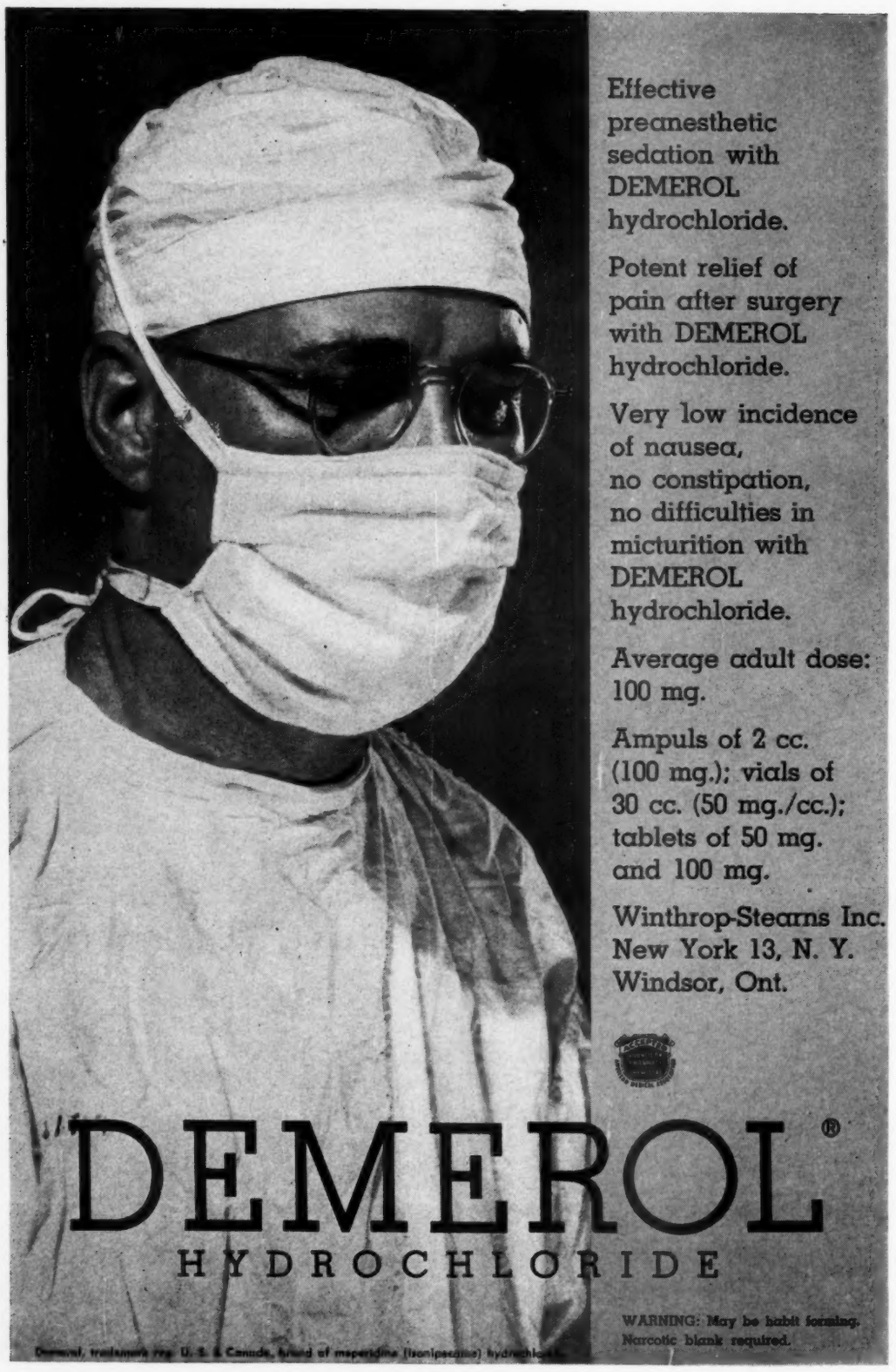

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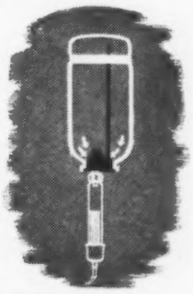

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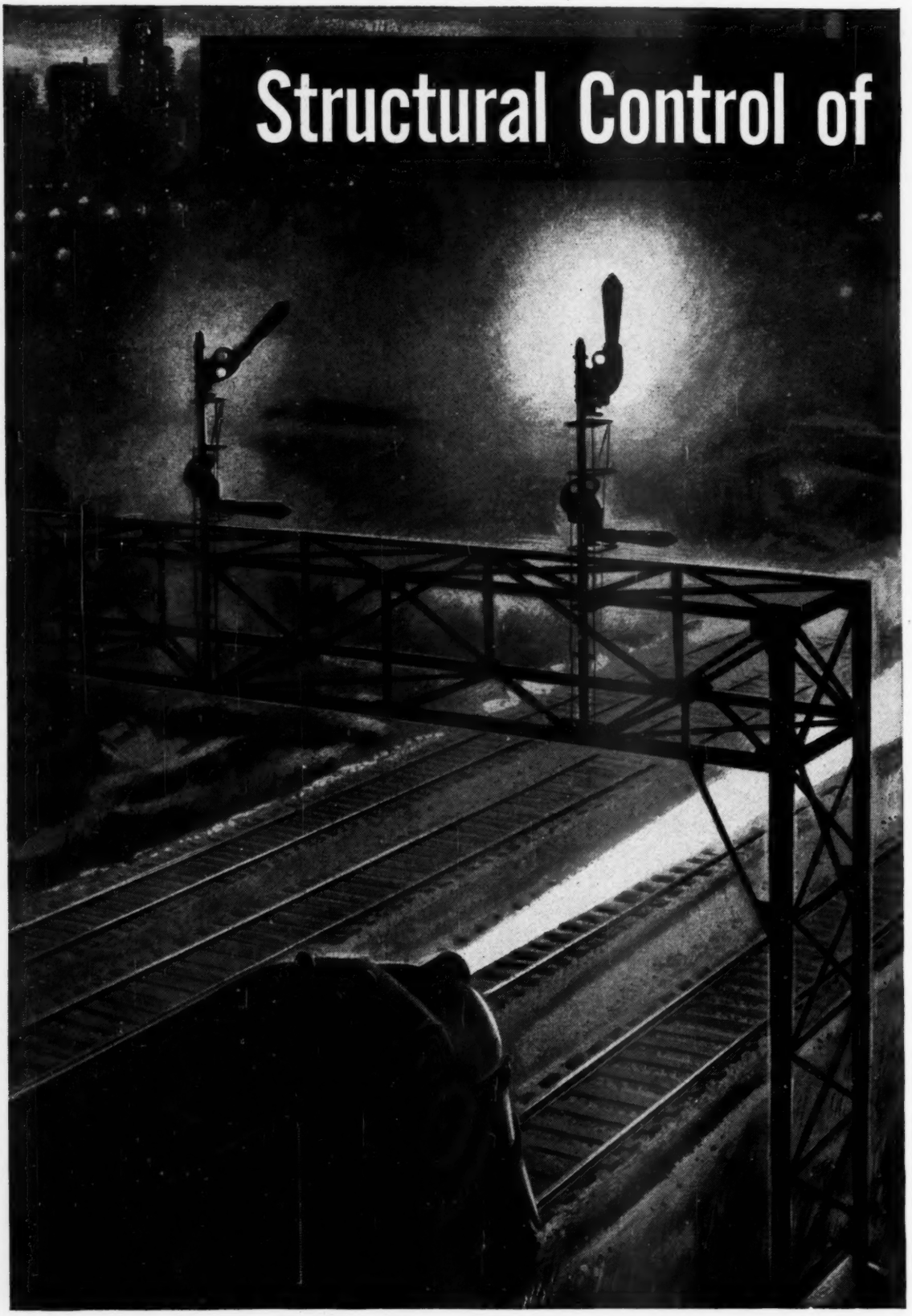
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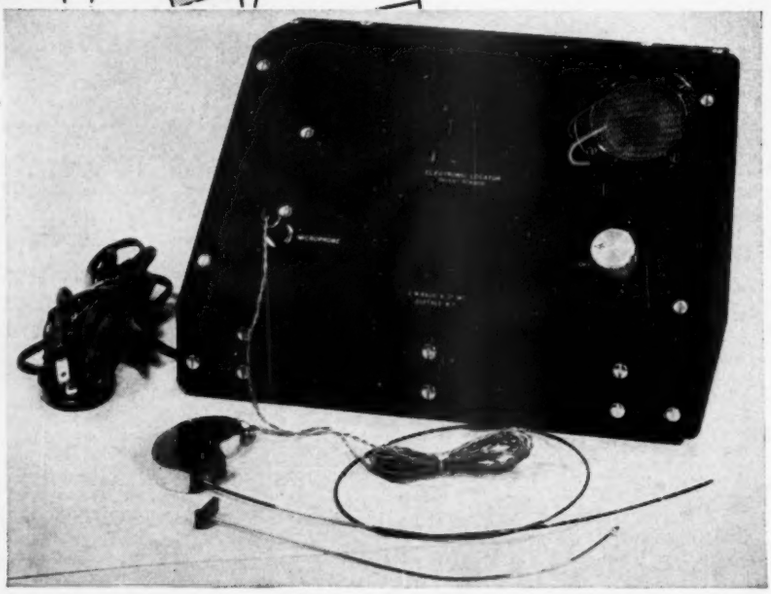

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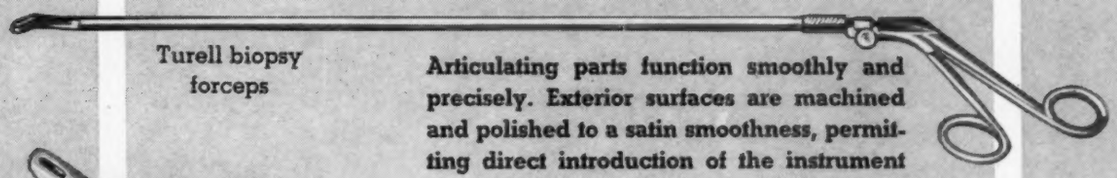

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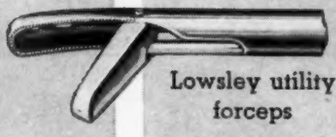

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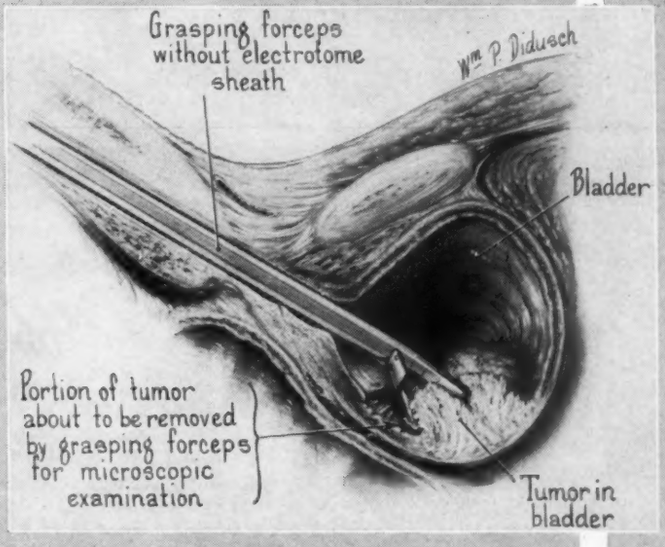
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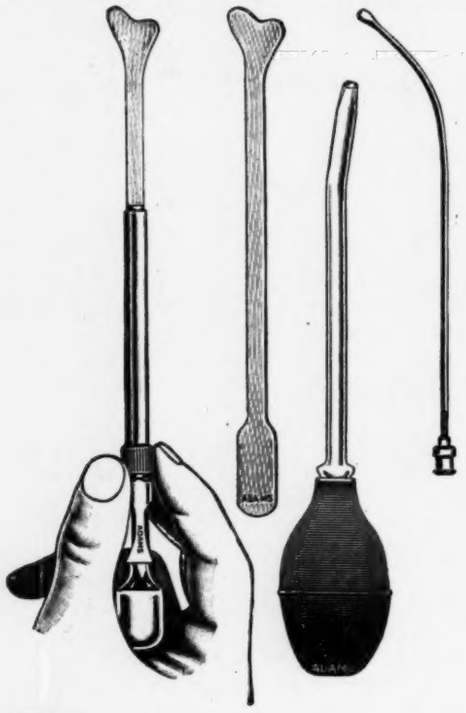

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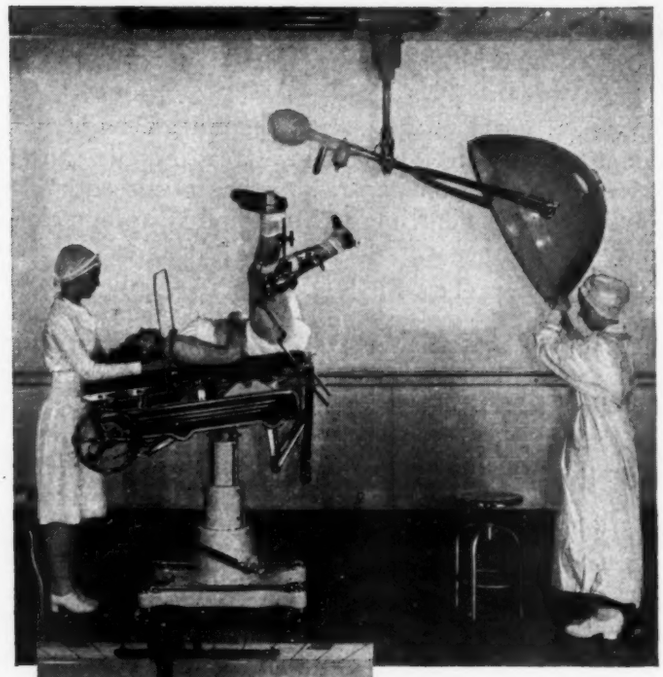

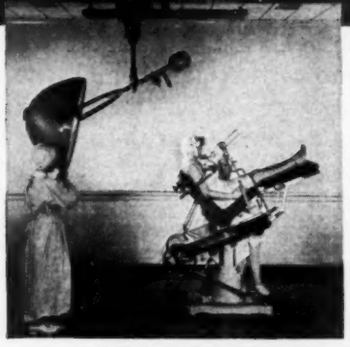

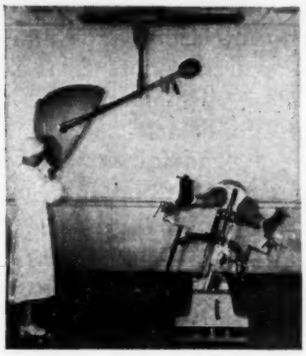

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(*continued on page* 28)

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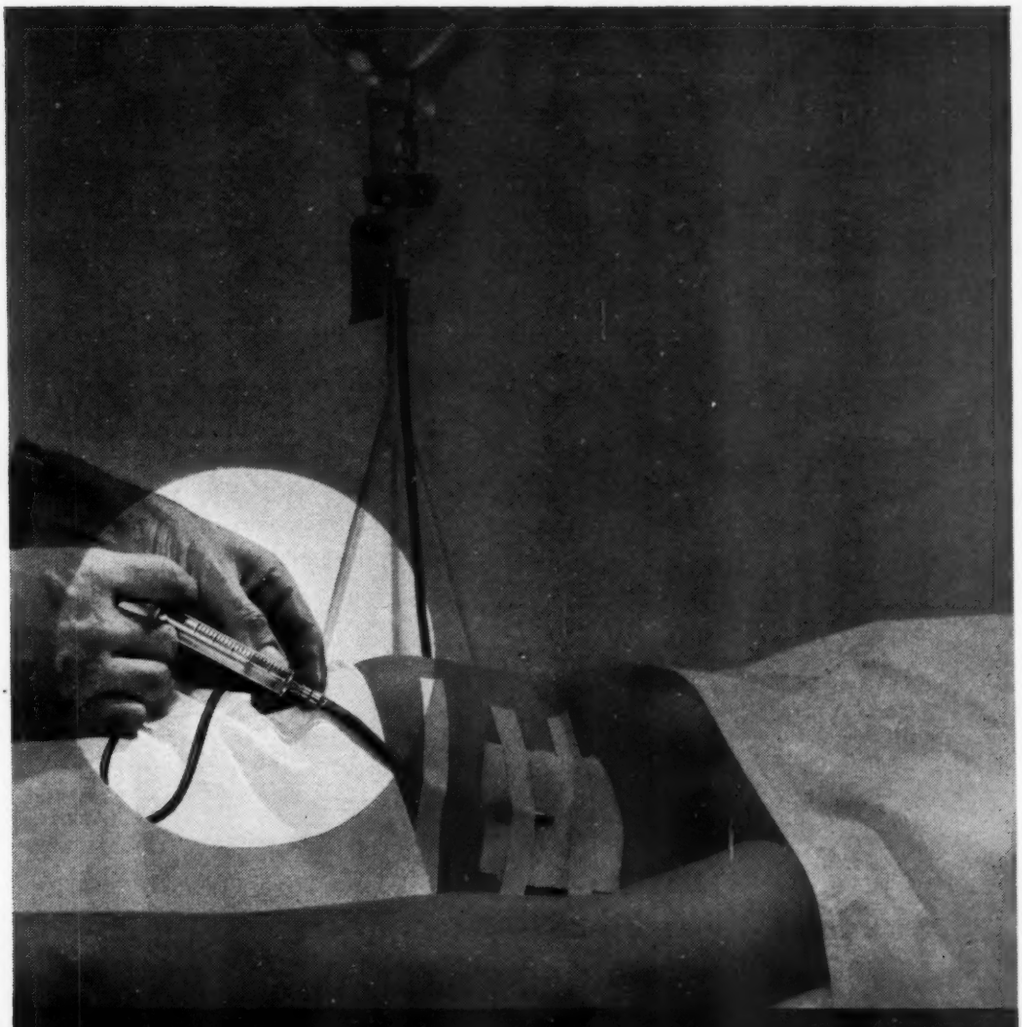

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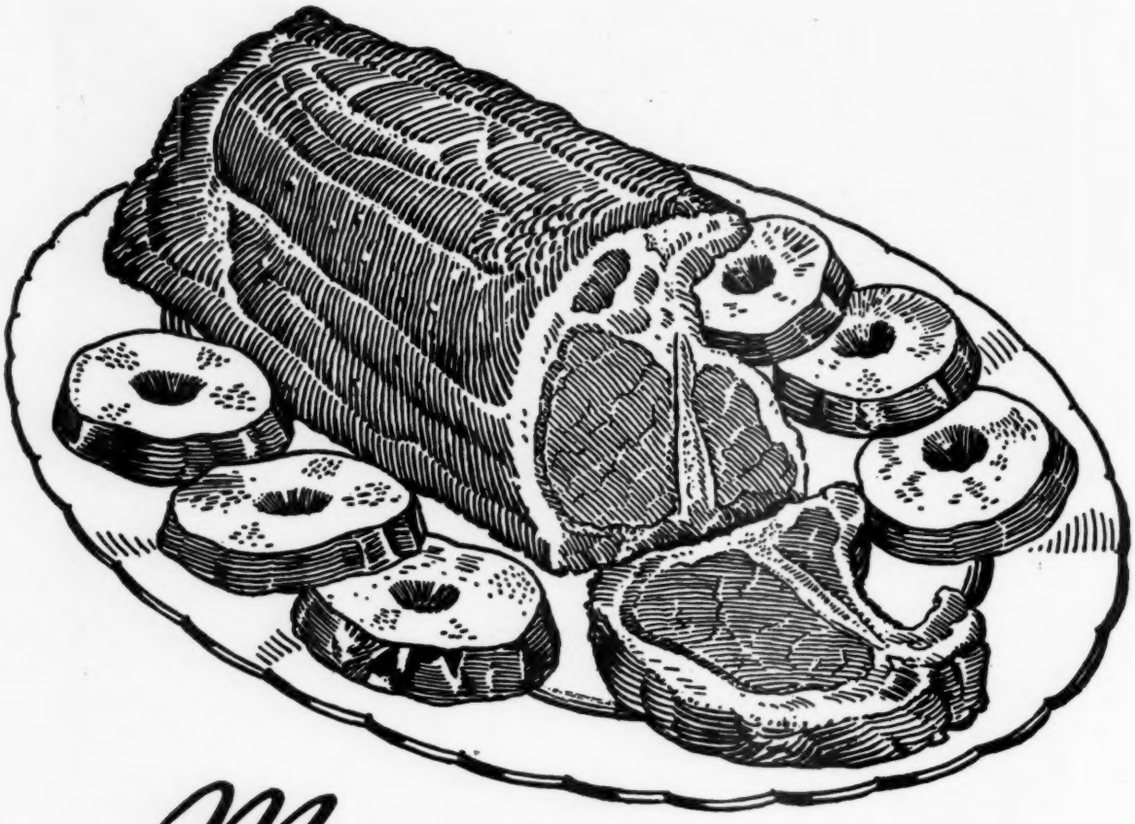

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*McLester, J. S.: Protein Comes Into Its Own, J.A.M.A. *139*:897 (Apr. 2,) 1949

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REFERENCES: 1. Organe, G.: *Lancet*, 1:773, 1949. 2. Hobson, J. A., and Prescott, F.: *Lancet*, 1:819, 1949. 3. Grob, D., Holaday, D. A., and Harvey, A. McG.: In Press. 4. Dripps, R. D.: *Jrl. Anesthesiology* In Press. 5. Organe, G., Paton, W. D. M., and Zaimis, E. J.: *Lancet*, 1:21, 1949. 6. Hewer, A. J. H., Lucas, B. G. B., Prescott, F., and Rowbotham, E. S.: *Lancet*, 1:817, 1949. 7. Castillo, J. C., and de Beer, E. J.: *Federation Proc.* Part I. 8:279, 1949; *J. Pharmacol. & Exper. Therap.* In Press. 8. Davies, D. L., and Lewis, A.: *Lancet*, 1:775, 1949.

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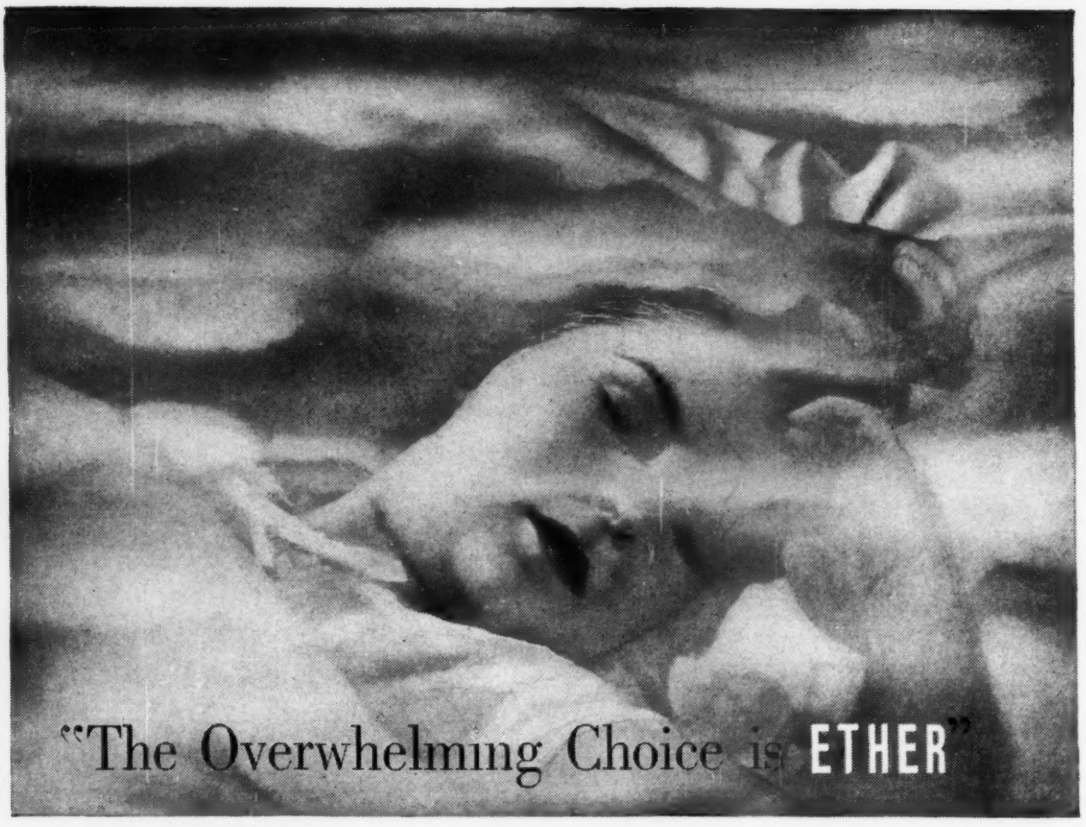

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*Burgoyne, J. R.: Surgery of the mouth and jaws, Dent. Items of Interest, 68: 479-89 (May) 1946.

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VOL. LXXVIII DECEMBER, 1949 NUMBER SIX

Editorial

PERITONITIS IN THE AGED

ELDERLY patients should not be condemned to die because they are judged too old for surgical treatment. Complications such as peritonitis to which aged patients are subject differ only in degree from those of others. However, more careful preoperative study is required taking into account the physiologic state of the body and extrinsic factors affecting the body's degenerative processes rather than the chronologic age.

In the aged the progress of infection is hastened by enfeebled circulation, senile emphysema, atrophy of lymphoid tissue and decrease in the formation of antibodies. The general resistance of the aged is depressed by arteriosclerotic changes, cardiac diseases, numerous nutritional disorders, local conditions, such as prostatic hypertrophy and a despondent mental attitude toward life.

Peritonitis causes death in the aged in the same manner as it does in the younger group and this is either by profound or overwhelming toxemia or by chemical and nutritional changes which accompany the associated small intestinal obstruction. If persistent leakage from a hollow viscus does not exist and if toxemia and obstruction can be controlled, the death rate could be maintained at a minimum.

Peritonitis can be grouped into three classes: the first due to perforations of the gastrointestinal tract thereby including lesions of the gallbladder, stomach and intestine; the second group due to spread of a mixed infection from a primary source such as an acute suppurative appendicitis; and the third group due to rapidly spreading invasion of the peritoneum by virulent organisms.

In the first group if there is persistent leakage from a perforated viscus, the patient will not survive unless the leakage is closed within a reasonable period of time. The presence of gas in the peritoneal cavity will give conclusive evidence of the presence of a perforation. Unless the perforation appears to be sealing off and the peritonitis localized, or unless the leakage such as in a perforated peptic ulcer can be controlled with continuous gastric suction, the indications for surgical intervention are clear.

In the second group if the general condition of the patient is good, leakage of a perforated appendix is in most cases never progressive. The problem is then control of a localized infection by organisms which are apt to be virulent and resistant to antibiotics. However, with advancing years the structure of the appendix changes, the lymphoid tissue has largely disappeared and fibrous atrophic changes associated with vascular changes are present that cause an inability of the circulation to

respond to the demands of inflammation. Hence the devitalized appendix subject to the obstruction of its lumen by a fecolith soon perforates and, as the omentum and peritoneum frequently lack ability to localize the infection, general peritonitis usually ensues without classic signs or symptoms.

As for the third group, that is peritonitis caused by pneumococcus or streptococcus, the condition responds to chemotherapy and antibiotic agents so quickly that the final outcome is uneventful in most patients.

Postoperative peritonitis presents other problems. In these patients it is necessary to decide whether infection is due to leakage or the result of contamination. The problem is rendered more difficult because the presence of air in the peritoneal cavity is expected and does not help to establish the diagnosis of perforation. Secondary operations except for simple drainage or possibly ileostomy are rarely undertaken in these patients because it is often quite difficult to establish the diagnosis. One has to resort to gastrointestinal decompression, antibiotics, chemotherapy and general supportive measures.

Generally speaking there is still no specific treatment for acute peritonitis. The successful outcome is dependent on early diagnosis with prompt and judicious surgical intervention aided by chemotherapy and judicious preoperative care.

Preoperatively some consideration should be given to the maintenance of nutritional balance since the store of essential food substance is low; particular attention should be given to blood proteins, protein maintenance and vitamins. Cardiac incompetence should be studied and reserve in tissue functions carefully considered. The anesthetic should be chosen which will produce minimal visceral damage and I firmly believe inhalation anesthesia should be avoided whenever possible. The operation should not be unduly long and whenever possible minimal surgery should be done; however, one should not hesitate to carry out the proper surgical procedure when indicated simply because of the individual's age. One must always bear in mind that greater gentleness is necessary in these patients since their tissue is much more easily injured by minor trauma.

Intestinal obstruction has been perhaps the most important contributory factor in causing death from peritonitis. The Miller-Abbott tube has been of great help in attempting to overcome this factor. The postoperative abdominal distention with loss of large quantities of fluid and electrolytes into and from the gastrointestinal tract, disturbances in nutrition, protein metabolism and in acid base equilibrium are all potent factors in bringing about the unfavorable outcome. Today many of these factors can be prevented or controlled and obstruction should be a rare cause of death.

The two most important considerations in passing a Miller-Abbott tube are the foresightedness of the attending surgeon and perseverance of the house surgeon to whom details are entrusted. The time for passing such a tube is also important. If the tube is passed before operation and intestinal intubation is instituted successfully, one of the major problems of peritonitis is under control. If it is delayed until the patient is greatly distended, too ill to cooperate and the ileus is so far advanced that the bowel has lost its tone, the tube may be difficult to pass and the effects of intubation will be doubtful.

Fluid and electrolyte balance is a problem that requires serious consideration. If the plasma proteins and cardiac reserve are normal, there is little danger of giving enough fluid to cause cardiac decompensation or pulmonary edema; however, one must always keep in mind that the vessels of old people respond less rapidly and readily to changing conditions. If the urinary output is about 1,500 cc. or more a day and if the patient does not have chronic nephritis, there is little danger of giving too much salt unless all fluids are given in saline. Administration of excessive amounts of saline causes retention of salt with consequent retention of water and

disturbance in acid base equilibrium. Any blood chloride deficiency should be corrected promptly.

The frequency with which hypoproteinemia occurs in the aged has been demonstrated repeatedly. The condition may exist in the absence of edema. Even minor degrees of hepatic damage may be reflected in lowered levels of blood proteins. Many investigators have called attention to the important part that protein metabolism, particularly hypoproteinemia, plays in wound healing, predisposition to shock, postoperative edema in wounds, increased susceptibility to infection and hepatic dysfunction. It is therefore essential that an attempt be made preoperatively to increase protein reserves and postoperatively to maintain a positive nitrogen balance.

As for the control of infection it was not until the introduction of sulfonamides and antibiotics that we had any specific means of controlling pyogenic infections. Penicillin when used in large amounts is effective in controlling peritonitis due to mixed infection. By giving enough penicillin over a long period of time eventually more penicillin is given than the Escherichia coli can destroy or inactivate and finally a sufficient concentration of active penicillin is built up to inhibit the growth of the offending virulent cocci.

Sulfanilamides when administered in effective doses are valuable adjuncts in the treatment of these patients. They are used intravenously until tolerated by mouth and at all times should be used in combination with penicillin and streptomycin.

Clinical experience and experimental data indicate that the use of streptomycin is not as effective as the use of penicillin, particularly when the latter is used in combination with sulfadiazine. Streptomycin appears to be of the greatest value in the treatment of early spreading peritonitis. It should be used early, and in combination with penicillin and sulfadiazine.

Reports on the use of aureomycin appear favorable, however, this agent is still in the investigative stage.

If one reviews the usual cases of peritonitis in the aged it will be found that the mortality rate had been exceedingly high before the advent of the sulfa drugs and antibiotics. One will also note that mortality is still high in spite of the use of newer drugs. An analysis of the poor results will disclose the following: (1) Operations on seriously ill individuals are usually undertaken too late. (2) Operations are not performed because clinical signs of peritonitis found in these patients are not classic. (3) The general preoperative condition of older patients is not usually very good. (4) The elective removal of a diseased gallbladder or diseased appendix will sometimes prevent the possibility of peritonitis subsequently due to an acute cholecystis or acute appendicitis. (5) Exploratory laparotomies should be done more frequently on the equivical case of suspected intra-abdominal infection in the aged. (6) Surgical consultation should be sought early in any case of suspected intra-abdominal infection in an old person.

In general it may be stated that surgical aggressiveness should be encouraged in the management of these cases and successful management can be hoped for, I reiterate, only if (1) early diagnosis is made; this is at times most difficult in the aged because the signs and symptoms are not classic; (2) early surgical intervention is undertaken, with careful preoperative and postoperative care; (3) effective surgical intervention is undertaken; by this is implied that while the surgery be minimal it should also be definitive in spite of the age of the patient and (4) effective chemotherapy and adequate supportive measures are used.

THOMAS C. CASE, M.D.

Original Articles

MECKEL'S DIVERTICULUM*

A REVIEW OF THIRTY-NINE CASES

GEORGE P. SCHWEI, M.D. AND ARNOLD S. JACKSON, M.D.
Madison, Wisconsin

IN reviewing the literature on Meckel's diverticulum some interesting facts concerning its historical aspects are found. One author states that Littré in 1700 described a structure that was probably a true congenital diverticulum. Another states this was reported by Littré in 1742 which would be sixteen years after his death. One author reports Ruysh as observing this anomaly in 1701 yet another states, with a variation in spelling, that a Ruish made this observation in 1707. However, the literature goes on to tell us that Meckel was the first to publish an adequate description of this anomaly in 1809, 1812 or was it 1815? Some observers mention a Berlin anatomist, Johann Fredrick Meckel, but he died in 1777. Others mention a Johann Fredrick Meckel of Leipzig who was born in 1781 and, as near as can be figured out, must have been the grandson. At any rate the persistence of the duct connecting the midgut with the yolk sac in early embryonic life may lead to the formation of what is known as Meckel's diverticulum.

The literature on this subject has been very extensive, particularly during the late twenties and early thirties. During the past several years there seems to have been a revival of interest in this important anomaly, particularly because of the surprisingly high percentage of Meckel's diverticula showing serious pathologic complications. Then, too, it is said to be the most common anomaly of the entire intestinal tract.[6] Too many cases (2 to 3 per cent) have been reported at routine postmortem examinations and not enough at the time of surgery. We agree with Umphrey, who reported Meckel's diverticulum occurring only nine times in 3,460 intra-abdominal operations, that many cases obviously have been missed. Balfour reported only fifteen Meckel's diverticula in 11,107 abdominal operations. McGlauman reported only three cases in 14,000 laporatomies. In our own series of thirty-nine cases covering thirty-three years seventeen of the cases were found during the past ten years and during 1948. When an increased effort was made to uncover some of these cases at surgery, seven were found. It is the purpose of this paper to present a review of the thirty-nine cases encountered at the Jackson Clinic. Particular emphasis will be placed on the pathologic complications and also on the importance of carefully searching for this anomaly at intra-abdominal operations.

Age and Sex Incidence. In our series Meckel's diverticulum occurred twice as often in males as in females (twenty-six and thirteen) which is in agreement with most observers. Interestingly enough six of seven of the 1948 cases occurred in females. Possibly in the future the sex incidence will be more equally divided.

Excluding the youngest patient, age ten months, and the oldest, age seventy-two, the average age was twenty-five years. The age group fourteen to twenty-one represents the most cases (ten) followed by the seven to fourteen group (eight).

* From the Jackson Clinic, Madison, Wis.

Pathologic Complications. Forty-six per cent of the diverticula were incidental, that is, without disease. Fifty-three per cent or twenty-one cases showed pathologic changes. This incidence is considerably higher than that generally reported in the literature. We have grouped the different types of pathologic conditions according to Haber's classification. (Table 1.)

INFLAMMATORY TYPE

The preoperative diagnosis of Meckel's diverticulum is rarely made because the symptoms vary according to the type of diverticulum found. Also, acute diverticulitis is said to be rare because usually the opening of the diverticulum in the ileum is large, the contents liquid and the contents will empty simultaneously with the ileum. In this series, however, eleven cases showed inflammatory changes, seven being acute or gangrenous. Of these, three diverticula perforated with resulting peritonitis. Symptoms of this type usually resemble those of an inflamed viscus or a perforated viscus and simulate acute appendicitis or a perforated appendix.

CASE REPORTS

Case 1. No. 78136, a male, age fifty-one, was admitted to the hospital with the chief complaint of pain in the abdomen. The onset of the pain had been twenty-three hours prior to his admission and had begun as a sharp, knife-like pain in the right scrotum. The patient was unable to continue his chores about the farm because of the pain which caused him to double up. He felt nauseated but did not vomit. In lying down the pain became less severe. The patient spent a fairly comfortable night and in the morning noticed a dull, aching pain in the right lower quadrant. He attempted to milk a cow and the pain became worse, so the patient sought medical aid. There had been no previous episode of this nature but twelve years before the patient stated he had a large amount of intestinal bleeding and was treated by his local doctor. He received a blood transfusion and was confined to bed at home for three months because of weakness.

Physical examination revealed a moderately dehydrated, acutely ill patient in apparent distress. The head, neck, heart and lungs were essentially normal. The abdomen was moderately distended with tenderness most marked in the right lower quadrant. No masses were present. His temperature was 100.5°F., pulse 100 and respirations 28; blood pressure was 108/80. The hemoglobin was 80 per cent and the white blood count 17,950. A preoperative diagnosis of acute appendicitis was made.

The abdomen was explored under spinal anesthesia through a right pararectus incision. On opening the peritoneum a lemon yellow exudate was encountered. The loops of small bowel were densely adherent and it was impossible to identify the cecum or colon. The entire intestines were covered with a thick, chronic, inflammatory caul. Dissection of the main inflammatory mass revealed a ruptured Meckel's diverticulum. This was resected and the abdomen was drained and closed in layers. The patient's postoperative convalescence was stormy for the first six days and then improved. He was discharged on the twenty-first day.

It was thought that this patient apparently had a massive hemorrhage and widespread peritonitis from the Meckel's diverticulum twelve years before and on this admission suffered from an acute diverticulitis with perforation and another generalized peritonitis.

Case 11. No. 48280, a female, age seventy-two, complaining of severe generalized abdominal pain and vomiting of two days' duration, was brought to the hospital by her local doctor. Two days before admission she was up and about doing her housework when she was suddenly seized with a stabbing abdominal pain followed by vomiting. The pain remained constant and appeared to be located in both lower quadrants. There had been no previous acute attack but the patient stated she had abdominal cramps with occasional vomiting and constipation for about ten years.

Physical examination revealed a markedly dehydrated, acutely ill patient with an ashen gray complexion; she appeared toxic. Her temperature was 101.6°F., pulse 120 and respirations 28. The white blood count was 18,000. Physical signs and symptoms were limited to the abdomen which was greatly distended, rigid and diffusely tender throughout. Pelvic examination revealed a large, tender, fluctuating mass in the posterior cul-de-sac. A preoperative diagnosis of pelvic abscess due to a ruptured appendix was made.

TABLE 1

Case	Age	Sex	Year	Abdominal Pain	Vomiting	Melena	Hemoglobin (Per cent)	Duration of Symptoms	Preoperative Diagnosis	Postoperative Diagnosis	Pathologic Findings	Treatment	End Result
18071	10	M	1915	Yes	Yes	Yes	...	3 yr.	Intestinal hemorrhage	Meckel's diverticulum with perforating ulcer	Meckel's diverticulum inflamed 20 inches from ileocecal valve; perforating ulcer	Resection of bowel with side-to-side anastomosis	Recovery
14845	48	M	1916	Yes	No	No	...	6 mo.	Acute appendicitis	Appendicitis and acute diverticulitis	Appendicitis and acute diverticulitis	Appendectomy and resection of diverticulum	Recovery
21845	19	M	1920	Yes	No	No	85	8 mo.	Chronic appendicitis	Acute Meckel's diverticulum	Acute diverticulitis	Appendectomy and resection of diverticulum	Recovery
29602	25	M	1921	Yes	No	No	...	6 mo.	Chronic appendicitis	Same with Meckel's diverticulum	Retrocecal appendix. Meckel's diverticulum 18 in. from ileocecal valve	Appendectomy and resection of diverticulum	Recovery
34321	25	M	1923	Yes	Yes	No	...	3 da.	Intestinal obstruction	Same due to Meckel's diverticulum	Normal appendix; Meckel's diverticulum 30 in. from ileocecal valve and adherent to proximal bowel by adhesions	Bowel freed and raw surfaces covered with omentum graft; Meckel's diverticulum not removed	Recovery
34693	44	M	1924	Yes	No	No	85	4 mo.	Chronic appendicitis	Same with Meckel's diverticulum	Appendix subacutely inflamed; tip of Meckel's diverticulum acutely inflamed	Appendectomy and resection of Meckel's diverticulum	Recovery
48072	14	F	1926	Yes	Nausea	No	80	7 da.	Subacute appendicitis	Subacutely inflamed Meckel's diverticulum	Meckel's diverticulum subacutely inflamed 18 in. from ileocecal valve; appendix normal	Appendectomy and resection of Meckel's diverticulum	Recovery
48280	72	F	1926	Yes	Yes	No	...	10 da.	Pelvic abscess due to ruptured appendix	Generalized peritonitis due to ruptured appendix	Rupture of base of Meckel's diverticulum with abscess formation and peritonitis	Resection of Meckel's diverticulum and drainage of abdominal cavity	Death (19th postoperative day)
49850	14	M	1926	Yes	No	Yes	15	4 da.	Intestinal hemorrhage due to Meckel's diverticulum	Same	Meckel's diverticulum 16 in. from ileocecal valve with bleeding ulcer	Appendectomy and resection of Meckel's diverticulum	Recovery
53276	27	M	1927	Yes	No	No	90	14 da.	Chronic appendicitis	Same with Meckel's diverticulum	Subacute appendix and non-inflammatory Meckel's diverticulum	Appendectomy with resection of Meckel's diverticulum	Recovery
37895	52	M	1929	Yes	No	No	85	1 yr.	Chronic appendicitis	Subacutely inflamed Meckel's diverticulum	Meckel's diverticulum 18 in. from ileocecal valve; non-inflammatory	Appendectomy with resection of Meckel's diverticulum	Recovery
58631	29	M	1929	Yes	Yes	No	88	3 da.	Acute appendicitis	Same with Meckel's diverticulum	Acute appendix; Meckel's diverticulum 12 in. from ileocecal valve non-inflammatory	Appendectomy with resection of Meckel's diverticulum	Recovery
45165	28	F	1930	Yes	No	No	87	25 da.	Chronic appendicitis	Same with Meckel's diverticulum	Appendix subacutely inflamed; Meckel's diverticulum not inflamed	Appendectomy with resection of Meckel's diverticulum	Recovery
70524	33	M	1931	Yes	No	Yes	65	3 yr.	Intestinal hemorrhage due to Meckel's diverticulum	Same	Meckel's diverticulum 20 in. from ileocecal valve with ulceration	Resection of 9 in. bowel with side-to-side anastomosis	Recovery
69229	18	M	1931	Yes	No	No	97	14 da.	Acute appendicitis	Subacute appendicitis; subacute diverticulitis	Same	Appendectomy and resection of Meckel's diverticulum	Recovery
78136	51	M	1933	Yes	No	Yes (12 yr. ago)	...	1 da.	Acute appendicitis	Ruptured Meckel's diverticulum with generalized peritonitis	Ruptured Meckel's diverticulum with generalized peritonitis and dense small bowel adhesions	Resection of Meckel's diverticulum and drainage	Recovery
76125	10 mo.	M	1933	Yes	Yes	No	...	1 da.	Intestinal obstruction	Intestinal obstruction due to Meckel's diverticulum	Tip of Meckel's diverticulum adherent to lower iliac mesentery; 18 in. of ileum were gangrenous	Excision of Meckel's diverticulum and exteriorization of gangrenous bowel	Death (1st postoperative day)
65603	8	M	1933	Yes	Yes	No	80	1 da.	Acute appendicitis with early peritonitis	Ruptured Meckel's diverticulum and generalized peritonitis	Gangrenous Meckel's diverticulum with perforation and generalized peritonitis	Appendectomy; resection of Meckel's diverticulum and abdominal drainage	Recovery
72750	33	M	1933	Yes	No	No	...	1 yr.	Chronic appendicitis	Same with Meckel's diverticulum	Retrocecal appendix; Meckel's diverticulum not inflamed	Appendectomy and resection of Meckel's diverticulum	Recovery
83114	21	M	1935	Yes	Yes	No	...	21 da.	Acute appendicitis	Acute Meckel's diverticulum; acute mesenteric lymphadenitis	Acutely inflamed Meckel's diverticulum 13 in. from ileocecal valve; mesenteric glands enlarged	Appendectomy and resection of Meckel's diverticulum	Recovery

84735	12	F	1935	Yes	No	No	...	1 yr.	Subacute appendicitis	Same with Meckel's diverticulum	Appendix subacutely inflamed; Meckel's diverticulum 13 in. from ileocecal valve; mesenteric glands enlarged	Appendectomy; Meckel's diverticulum not removed	Recovery
89688	12	M	1937	Yes	Yes	Yes	30	15 mo.	Intestinal hemorrhage due to Meckel's diverticulum	Same	Meckel's diverticulum 24 in. from ileocecal valve with bleeding ulcer at tip	Appendectomy; Meckel's diverticulum not removed	Recovery
73180	19	M	1938	Yes	Yes	No	...	1 da.	Strangulated incisional hernia	Incarcerated incisional hernia; Meckel's diverticulum	Omental and small bowel incarceration; Meckel's diverticulum not inflamed	Adhesions freed; fascia repaired; Meckel's diverticulum not removed	Recovery
80721	43	M	1938	Yes	No	No	70	1 yr.	Chronic cholecystitis with cholelithiasis	Same with Meckel's diverticulum	Meckel's diverticulum not inflamed	Cholecystectomy; Meckel's diverticulum not removed	Recovery
93044	20	F	1938	Yes	Yes	No	78	1 yr.	Subacute appendicitis	Same with Meckel's diverticulum	Appendix subacutely inflamed; fecoliths; Meckel's diverticulum 12 in. from ileocecal valve; not inflamed	Appendectomy with resection of Meckel's diverticulum	Recovery
100179	24	F	1940	Yes	No	No	103	21 da.	Chronic appendicitis	Same with Meckel's diverticulum	Chronic appendicitis; Meckel's diverticulum not inflamed	Appendectomy; resection of Meckel's diverticulum	Recovery
108849	36	M	1942	Yes	No	No	...	1 da.	Acute appendicitis	Subacute appendicitis and Meckel's diverticulum	Subacute appendix; Meckel's diverticulum 15 in. from ileocecal valve; not inflamed	Appendectomy; Meckel's diverticulum not removed	Recovery
109009	19	M	1942	Yes	No	No	...	1 yr.	Subacute appendicitis	Same with Meckel's diverticulum	Appendix subacutely inflamed; Meckel's diverticulum subacutely inflamed	Appendectomy with resection of Meckel's diverticulum	Recovery
99710	16	M	1942	Yes	Yes	No	...	30 da.	Intestinal obstruction due to postoperative adhesions; appendectomy done elsewhere 2 weeks before	Intestinal obstruction due to Meckel's diverticulum	Small bowel obstruction due to volvulus caused by Meckel's diverticulum attached to umbilicus	Volvulus reduced; adhesions freed; resection of Meckel's diverticulum	Recovery
117318	32	M	1943	Yes	No	Yes	80	Melena 16 yr. before	Intestinal hemorrhage due to Meckel's diverticulum	Same	Meckel's diverticulum 12 in. from ileocecal valve; marked scarring and induration with 1½ mm. ulcer at base	Resection of 5 in. of ileum and side-to-side anastomosis	Recovery
127014	8	F	1945	Yes	No	No	80	6 da.	Acute appendicitis	Mesenteric lymphadenitis and Meckel's diverticulum	Normal appendix; enlarged mesenteric glands; Meckel's diverticulum 10 in. from ileocecal valve not inflamed	Appendectomy and resection of Meckel's diverticulum	Recovery
126552	55	M	1947	Yes	No	No	...	2 mo.	Chronic cholecystitis	Same and Meckel's diverticulum	Chronic cholecystitis with lithiasis; Meckel's diverticulum 18 in. from ileocecal valve; not inflamed	Cholecystectomy and resection of Meckel's diverticulum	Recovery
69439	23	M	1948	Yes	Yes	No	90	1 da.	Acute appendicitis	Meckel's diverticulum 15 in. from ileocecal valve subacutely inflamed	Meckel's diverticulum subacutely inflamed; appendix normal	Appendectomy and resection of Meckel's diverticulum	Recovery
69530	17	F	1948	Yes	No	No	90	2 da.	Acute appendicitis	Meckel's diverticulum 18 in. from ileocecal valve subacutely inflamed	Meckel's diverticulum subacutely inflamed; gastric mucosa	Appendectomy and resection of Meckel's diverticulum	Recovery
69624	30	F	1948	...	...	...	...	.....	Diastasis recti	Same and Meckel's diverticulum	Meckel's diverticulum 36 in. from ileocecal valve; not inflamed	Resection of Meckel's diverticulum	Recovery
71227	30	F	1948	Yes	Yes	No	88	3 yr.	Acute appendicitis	Mesenteric lymphadenitis and Meckel's diverticulum	Mesenteric glands enlarged; Meckel's diverticulum 30 in. from ileocecal valve; not inflamed	Appendectomy and resection of Meckel's diverticulum	Recovery
152941	21	F	1948	Yes	No	No	94	1 da.	Acute appendicitis	Same with Meckel's diverticulum	Acute appendix; Meckel's diverticulum 8 in. from ileocecal valve; not inflamed	Appendectomy and resection of Meckel's diverticulum	Recovery
136951	17	F	1948	Yes	No	No	80	1 da.	Acute appendicitis	Same and Meckel's diverticulum	Acute appendix; Meckel's diverticulum 18 in. from ileocecal valve; not inflamed	Appendectomy and resection of Meckel's diverticulum	
141474	18	F	1948	Yes	Yes	No	80	10 da.	Acute appendicitis	Subacute appendicitis and Meckel's diverticulum	Subacute appendix; Meckel's diverticulum 15 in. from ileocecal valve attached to umbilicus	Appendectomy andresection of Meckel's diverticulum	Recovery

1 2

FIG. 1. Photograph of surgically resected Meckel's diverticulum and appendix. Case No. 65603.
FIG. 2. Cut section revealing presence of enteroliths. Case No. 65603.

On the day of admission a posterior colpotomy under nitrous oxide was done which released a large amount of foul-smelling, purulent material. The patient showed some improvement during the following two weeks but her temperature continued to spike and the foul drainage became increasingly profuse. Fifteen days later the abdomen was explored under ether anesthesia through a lower right rectus incision. The lower ileum and sigmoid were plastered with a purulent exudate underlying which were rather dense adhesions. Freeing of the adhesions caused severe wall perforations of the sigmoid which were immediately sutured. In the left lower quadrant there was a large perforation of the base of a Meckel's diverticulum. The diverticulum was resected and the bowel was closed. The appendix was small and fibrosed. The abdomen was closed in layers and a cigarette drain inserted. The patient had a stormy postoperative course and gradually became weaker and weaker until she expired on the sixteenth postoperative day. The cause of death was generalized peritonitis due to a ruptured Meckel's diverticulum. No postmortem examination was obtainable.

CASE III. No. 65603, a boy, age eight, was admitted to the hospital complaining of generalized abdominal pain and vomiting. The night before admission the patient had eaten four apples before going to bed. Shortly thereafter he had severe abdominal cramps and could not sleep. Three hours later he became nauseated and vomited. He had several bouts of vomiting before his admission. The pain became less acute and on admission he complained of pain in both lower quadrants. Past history was non-contributory.

Physical examination revealed a fairly well hydrated but acutely ill patient. His temperature was 101.8°F., pulse 120 and respirations 32. The head, neck, lungs and heart were essentially normal. The abdomen was moderately distended with acute tenderness in both lower quadrants. Moderate rigidity was present in both lower quadrants but was more pronounced on the right. Rectal examination was nonrevealing. The white blood count was 18,200. A preoperative diagnosis of acute appendicitis with early peritonitis was made.

The abdomen was explored under spinal anesthesia through a small McBurney incision. Opening of the peritoneum revealed about 500 cc. of serosanguineous fluid lying free in the abdominal cavity. The appendix was congested and covered with a fibrinous exudate. A perforated Meckel's diverticulum was found 18 inches from the ileocolic valve. The diverticulum was approximately 4 inches long with an enlarged terminal bulbous end which contained many large enteroliths. The gangrenous portion of this end had perforated. The diverticulum was resected, an appendectomy done and the wound closed in layers with a penrose drain inserted to the fascia. (Figs. 1 and 2.) The patient's postoperative convalescence was uneventful and he was discharged on the eleventh postoperative day.

CASE IV. No. 83114, a boy, age seven, entered the hospital complaining of pain in the abdomen and vomiting. The onset of pain had been twenty-six hours prior to his admission and began with stomach cramps while he was

playing outdoors. He vomited about an hour later. Pain continued as a dull ache and the patient vomited several times throughout the night after the ingestion of water. His mother stated that two years before he had a similar but less severe attack.

Physical examination revealed an alert, well developed, well nourished boy who did not appear to be in acute distress. Temperature was 99°F., pulse 124 and respirations 20. The white blood count was 20,150. The head, neck, lungs and heart were without significant changes. The abdomen was flat but somewhat resistant. Palpation of the right lower quadrant revealed acute tenderness. Rectal examination was negative but high palpation on the right elicited tenderness. The preoperative diagnosis was acute appendicitis.

The abdomen was explored under spinal anesthesia through a right pararectus incision; a small amount of free, straw-colored fluid was encountered on opening the peritoneum. The appendix appeared to be normal. An acutely inflamed Meckel's diverticulum was found 13 inches from the ileocolic valve. The mesenteric glands were numerous and enlarged. An appendectomy and resection of the diverticulum were done and the abdomen was closed in layers. The patient made an uneventful recovery and was discharged on the fourteenth postoperative day.

OBSTRUCTIVE TYPE

The symptoms of the obstructive type are the same as those of any other bowel obstruction. Haber and others state that the most numerous pathologic changes resulting from Meckel's diverticulum are of the obstructive type. This has not been true in our series. Intestinal obstruction is brought about in a number of ways. There may be torsion of a volvulus, intussusception knotting around the intestine, bands and remnants of the ompholemesenteric ducts and vessels, and incarceration in a hernia. Haber diagrammatically shows the different types of obstruction and describes in detail five cases representing the types of small bowel obstruction due to Meckel's diverticulum. Meckel's diverticulum occurs occasionally in connection with inguinal, umbilical and femoral hernia. Probably 50 per cent or less are incarcerated. Patterson in a recent review of the literature reports twenty-one cases of incarceration of Meckel's diverticulum in a femoral hernia and adds another case for a total of twenty-two. Atwood has reviewed the literature on intussusception due to an invaginated Meckel's diverticulum and reports an additional case. Following is a review of two of the obstructive cases encountered in our series.

CASE V. No. 76125, a male infant, aged ten months, was admitted to the hospital with the history of apparent abdominal pain and vomiting fourteen hours prior to admission. The night before entering the hospital the baby appeared to be in perfect health. He romped about the floor and was entertaining visitors. At 11:00 P.M. the infant awoke crying violently. He vomited and preferred lying on his side doubled up. A local doctor was called who examined the baby and prescribed treatment. At this time the abdomen was slightly distended but not rigid. The next morning the infant was again seen at which time the abdomen was moderately distended. No abdominal mass was felt. A rectal examination revealed some formed stool present but no blood or mucus. The child was pale and listless and was brought to the hospital. A flat film of the abdomen revealed rather advanced distention of loops of small bowel in the left abdomen. The cecum and transverse colon did not appear distended. A 2-ounce enema of hydrogen peroxide and sodium bicarbonate was given. There was an immediate expulsive effort and a formed stool 4 to 5 inches in diameter was evacuated. The baby seemed to brighten up at once and looked better. Hot stupes and subcutaneous fluids were administered. Within the next two hours the abdominal distention increased, there was no passage of gas or stool per rectum and the baby again appeared pale and listless. Surgery was decided on at once. A preoperative diagnosis of intestinal obstruction probably due to Meckel's diverticulum was made.

Under spinal anesthesia (20 mg. novacain) the abdomen was explored through a right rectus incision. The terminal end of a Meckel's diverticulum was adherent to the lower iliac mesentery. Eighteen inches of terminal ileum from the ileocecal junction to the site of the Meckel's diverticulum was black and gan-

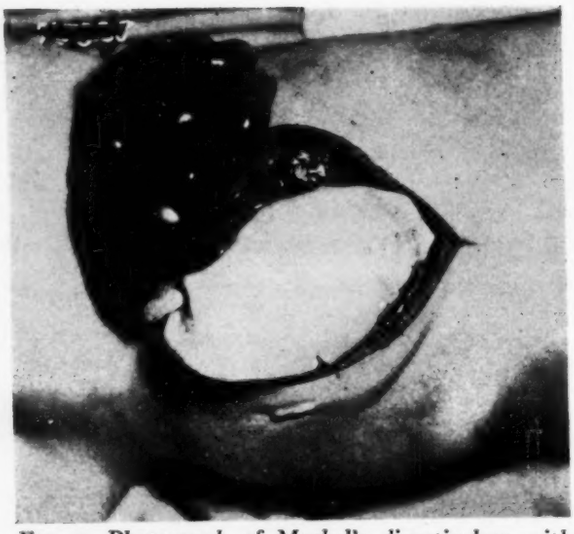

FIG. 3. Photograph of Meckel's diverticulum with loops of gangrenous bowel (post mortem). Case 76125.

grenous. (Fig. 3.) The diverticulum was excised, the gangrenous bowel with the cecum was exteriorized and the bowel was opened for drainage. The immediate postoperative condition of the infant appeared satisfactory. Four and a half hours later the infant suddenly expired. A postmortem examination which was limited to the operative incision only revealed what had been seen at surgery. It was believed, however, that death was due to a pulmonary embolus.

CASE VI. No. 99710, a boy, age sixteen, was admitted to the hospital complaining of abdominal pain and vomiting of three days' duration. Twelve days before his admission he had an appendectomy done elsewhere. His immediate postoperative convalescence was uneventful but on the tenth day his abdomen became distended, he vomited and had colicky pain. He had a small, difficult bowel movement. The vomiting continued on the eleventh and twelfth postoperative day and the parents stated the vomitus was dark brown with a fecal odor. There was no bowel movement the past two days.

Physical examination revealed an acutely ill sixteen year old boy who appeared markedly dehydrated. His temperature was 99°F., pulse 96 and respirations 28. The blood pressure was 140/90. The head, neck and chest showed no abnormality. The abdomen was distended and rigid. Tenderness was present in both upper quadrants; intestinal sounds were hyperactive and high-pitched. No masses were palpable. An open film of the abdomen showed a stepladder configuration of the small bowel indicating a low small bowel obstruction. The white blood count was 6,100, hemoglobin 112 per cent and red blood count 6,320,000. The urine specific gravity was 1,031, blood urea 84 mg. per cent and blood chlorides 375 mg. The patient was given intravenous fluids and was prepared for surgery. The preoperative diagnosis was small bowel obstruction due to postoperative adhesions.

The abdomen was explored under spinal anesthesia. The recent right rectus incision was reopened and slightly enlarged. On opening the peritoneum there was free escape of a moderate amount of free fluid transudate. Loops of small bowel were distended to about 2½ inches in diameter. The distended bowel was mahogany colored but not gangrenous. Peristalsis was visible below the point of obstruction which was 24 inches from the ileocecal valve. At this distance a large Meckel's diverticulum was found which remained attached to the umbilicus. A loop of ileum had become attached to the inflamed wall of the diverticulum and had rotated about the umbilical attachment causing the obstruction. Below the diverticulum the small bowel was adherent and kinked due to adhesions, causing further obstruction. The diverticulum was freed at its umbilical attachment and the volvulus untwisted. The adhesions were severed and the diverticulum was resected with the use of a Furniss clamp. A Wetzel type ileostomy was done and the abdomen was closed in layers. The patient ran a septic temperature for the first few postoperative days and then proceeded to complete recovery.

This case certainly emphasizes that any time a surgeon opens an abdomen and explores the lower quadrants for disorders in the absence of an acute infection he should examine at least 3 feet of terminal ileum and search for Meckel's diverticulum.

PEPTIC ULCERATION

The complications of peptic ulcer (ectopic gastric tissue) in Meckel's diverticulum is the same as in any other peptic ulcer, namely, bleeding and perforation. Intestinal hemorrhage is the most constant symptom. The blood may be either bright red, maroon or tarry but is usually maroon. Clots may be present but mucus is absent. Intestinal cramping should lead

one to suspect the diagnosis of Meckel's diverticulum. With minimal ulceration and the passage of an occasional tarry stool the diagnosis may be more difficult. Peptic ulceration in this anomaly is said to be an affliction of infancy or childhood. Messenger and Collins believe that no age group is immune. They report nine cases of intestinal hemorrhage, four of the patients being under four and a half years of age and five from seventeen to sixty years of age. Arnold Jackson in 1926 reported the first acute case of intestinal hemorrhage due to an ulcer of a Meckel's diverticulum. The patient was a boy, age fourteen, whose predominating symptom was the passing of bright red blood per rectum.

In the differential diagnosis of these cases intussusception must be ruled out. In intussusception a smaller amount of blood is passed and there is usually an admixture of blood and mucus. A mass can usually be palpated abdominally or by rectum. There is obvious evidence of intestinal cramping and pain occurs paroxysmally every few minutes.

Case VII. No. 49850,* a boy, aged fourteen, was admitted to the hospital with the chief complaint of repeated massive intestinal hemorrhage of four days' duration. The patient was seized with severe generalized abdominal cramps following the eating of green apples a few days previously. A physician was called who treated him for green apple colic. The boy was relieved of the pain with sedatives but became progressively weaker. On the third day considerable bright blood containing many clots was passed from the rectum. Another physician was called. He observed the further passage of several pints of blood varying in color from maroon to bright red. Noticing the marked anemia, progressive weakness and absence of general physical signs, except for slight tenderness in the right lower quadrant, he was vividly reminded of the clinical picture of a similar case he had seen two years previously and made a diagnosis of intestinal hemorrhage from Meckel's diverticulum.

On admission to the hospital physical examination revealed an acutely ill, very pale, sixteen year old boy who was in shock. His pulse was 128, very weak, temperature 100°F. and respirations 20. Hemoglobin was 12 per cent and the red blood count 1,210,000. The head, neck and chest showed no abnormality. The abdomen was flat, non-rigid and palpation elicited only slight tenderness in the right lower quadrant. The patient immediately received intravenous fluids and a whole blood transfusion of 500 cc. of citrated blood. The patient remained semi-comatose for two hours and gradually improved. The following day he again received a whole blood transfusion and his general condition improved greatly. Following further supportive measures the patient's blood picture continued to improve and he was up and around within a week. After the first twenty-four hours no further intestinal hemorrhages were seen. On the twelfth hospital day an exploratory operation was done.

The abdomen was explored through a subumbilical right rectus incision. On opening the peritoneum a small amount of straw-colored fluid was allowed to escape. The terminal ileum was located and about 40 cm. from the ileocecal valve a Meckel's diverticulum measuring 3 by 8 cm. was found. The terminal end was free and there was no visible or palpable evidence of hemorrhage. The appendix contained several fecal concretions and appeared edematous. No other pathologic lesions were found. The diverticulum was resected and an appendectomy was performed. The patient had an uneventful postoperative convalescence and was discharged on the tenth postoperative day.

Pathologic examination of the diverticulum showed the presence of the characteristic areas of gastric mucosa and an area of ulceration which was the site of the hemorrhage.

Case VIII. No. 70524, a male, aged thirty-three, was admitted to the hospital with the chief complaint of bloody stools. While in the service in France during World War I he had an attack of intestinal flu associated with severe vomiting and blood-tinged stools. Following this he was well until three years before admission when he noticed a few tarry stools. He was examined but nothing was found to account for his trouble. He was well until three weeks before admission when he had similar dark stools and felt faint. He had no pain or abdominal distress. The morning he entered the hospital he had a very large bloody stool estimated at about 1 quart.

* This case was previously reported by A. S. Jackson.

On admission to the hospital physical examination revealed a chronically ill, fairly well nourished white male. His temperature was 99.8°F., pulse 120 and respirations 22. The hemoglobin was 65 per cent, red blood cells 3,200,000, white blood cells 11,800, with 78 per cent neutrophiles and 20 per cent lymphocytes. The head, neck and chest showed no abnormality. The abdomen was flat and there were no palpable masses. Pressure over McBurney's point elicited moderate tenderness. Bowel sounds were normal; rectal examination was negative. A proctoscopic examination revealed the lumen to be filled with dark red clots as far as the reach of the proctoscope. At roentgenologic examination no lesion was found in the gastrointestinal tract. The patient received a blood transfusion and other supportive treatment and was operated upon on his third hospital day. A preoperative diagnosis of intestinal hemorrhage due to Meckel's diverticulum was made.

The abdomen was explored under spinal anesthesia through a lower right rectus incision. The entire colon was found to be filled with dark, clotted blood. A nodular mass was found 20 inches from the ileocecal valve. The mass together with 9 inches of ileum was resected and a side-to-side anastomosis was done. Upon opening the specimen it was found to be a large Meckel's diverticulum with a large ulcer crater at its base. The abdomen was closed in layers. The patient made a very uneventful recovery and was discharged on the fifteenth postoperative day.

Case IX. No. 117-318, a male, age thirty-two, was admitted to the hospital with the chief complaint of rectal bleeding. On the morning of his admission to the hospital he had a bowel movement and noticed bright red blood in the stool. The previous day he had experienced a stomach ache but no nausea or vomiting. He gave a history of previous rectal bleeding sixteen years before and again nine years before; neither of these episodes were serious enough to require hospitalization.

Physical examination revealed a well nourished, well developed male who did not appear acutely ill. His temperature, pulse and respirations were normal. The blood pressure was 140/60; hemoglobin was 80 per cent and the red blood count 4,200,000. Bleeding and coagulation time were normal. Examination of the head and neck was negative. There was a loud-blowing, precordial systolic murmur heard loudest at the apex of the heart. Examination of the abdomen was negative. Rectal examination revealed the presence of clotted blood. A proctoscopic examination merely indicated that the bleeding was higher in the gastrointestinal tract. The patient was given five blood transfusions in the next ten days. On his fifth hospital day during a transfusion the patient had another severe rectal hemorrhage. His hemoglobin dropped to 65 per cent and the red blood cells to 2,800,000. The patient was given further supportive treatment by the use of koagamin (oxalic acid). The patient gradually improved and was discharged on his fifteenth postoperative day. He was instructed to return in ten days for x-ray studies of the gastrointestinal tract. These roentgenograms were negative and an exploratory operation was advised. The preoperative diagnosis was intestinal hemorrhage due to Meckel's diverticulum.

The abdomen was explored under spinal anesthesia through a pararectus incision. The appendix was mildly inflamed and was removed. Twelve inches from the ileocecal valve a firm cicatricial mass was found in the ileum. Closer examination showed an hourglass type of Meckel's diverticulum which extended 1½ inches into the mesentery. Because of the induration and scar tissue surrounding the diverticulum about 5 inches of ileum were resected and a side-to-side anastomosis was performed. Exploration of other abdominal organs was negative. The abdomen was closed in layers.

Pathologic sections (Figs. 4 and 5) showed a diverticulum composed chiefly of a gastric type of mucosa. In the smaller part of the diverticulum a chronic ulcer extended to the muscularis mucosa. The patient had an uneventful postoperative course and was discharged on the fourteenth postoperative day.

Case X. No. 89688, a boy, aged twelve, entered the hospital complaining of the passage of bright red blood with loose bowel movements for the past four days. The patient and his mother stated that for the past fifteen months he had similar attacks at three- to four-month intervals. The attacks continued for three to four days and seemed to follow a definite sequence of events. The first day there would be a headache followed by vomiting; on the second day the passage of bright red rectal blood would be noticed with a semi-liquid

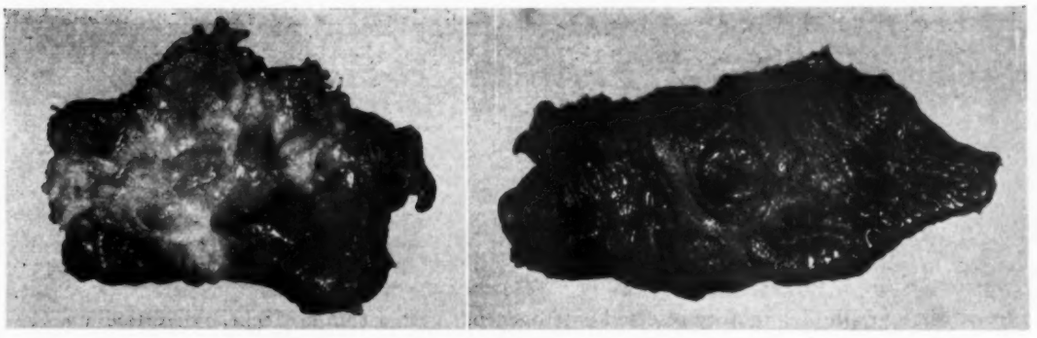

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FIG. 4. Photograph of surgically resected Meckel's diverticulum showing cut section of mesenteric portion. Case No. 117318.

FIG. 5. Same specimen showing mucosal surface with chronic ulceration through the muscularis mucosa. Case No. 117318.

stool. On the third day the stool would be dark and remain so for a few more days and then clear spontaneously. Abdominal pain was usually not a symptom.

Physical examination revealed a very anemic and chronically ill-appearing boy. His temperature was 100.4°F., pulse 140 and respirations 22. The blood pressure was 94/60; hemoglobin was 30 per cent, red blood count 1,810,000, white blood count 8,540, with 84 per cent neutrophiles and 14 per cent lymphocytes. Bleeding time was seven and a half minutes and coagulation time six minutes. The physical examination was entirely negative except for dark blood on the examining finger following a rectal examination. A proctoscopic examination was essentially negative except for the presence of black tarry fluid coming from above a 15 cm. level. The colon and small bowel on roentgenologic examination showed normal progress of the barium and no evidence of any pathologic lesions. The patient received several whole blood transfusions and other supportive treatment and was operated upon on his sixth hospital day. A preoperative diagnosis of intestinal hemorrhage due to Meckel's diverticulum was made.

Under spinal anesthesia the abdomen was explored through a right pararectus incision. The colon was filled with blood. Twenty-four inches from the ileocecal valve a Meckel's diverticulum measuring 3 by 1½ cm. was found. The base was indurated and on the mucosal surface an ulcer with evidence of recent hemorrhage was seen. (Fig. 6.) The diverticulum was resected at its base and the bowel closed transversely. The appendix appeared normal but was removed at the family's request. The wound was closed in layers. The postoperative course was uneventful and the patient was discharged on the twelfth postoperative day.

FIG. 6. Photograph of Meckel's diverticulum at operation prior to resection. Note ulcer with evidence of recent hemorrhage. Case No. 89688.

TUMORS IN MECKEL'S DIVERTICULUM

Various types of tumors or tumor tissue have been found in Meckel's diverticulum. Excluding gastric and pancreatic heterotopic tissue, if they may be classified as tumors, the incidence of benign or malignant tumors is very low. Tumors may form from the normal cellular elements of the intestinal mucosa or more frequently from the aberrant tissue elements of gastric and pancreatic mucosa. The gastric type has been found in 15 to 30 per cent of all cases. The pancreatic type is found less frequently.

The literature contains isolated reports of benign tumors which include adenomas, myomas, lipomas, papillomas, neuromas, enterogenous cysts, carcinoids, etc. Car-

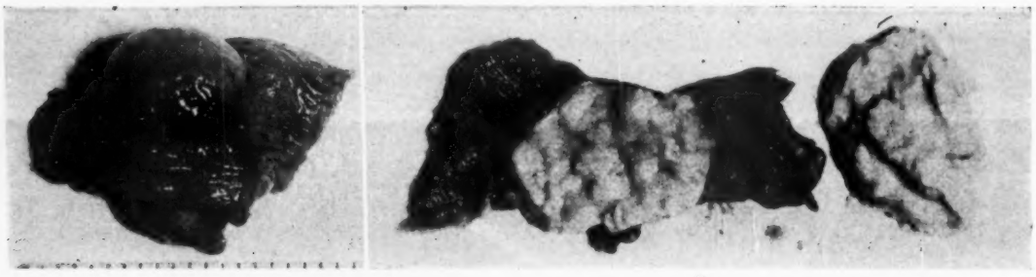

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FIG. 7. Photograph of surgically resected enterogeneous cyst within a Meckel's diverticulum. Case No. 93044.
FIG. 8. Cut section revealing tightly packed caseous material surrounded by chronic inflammatory changes.

cinoid or argentaffin tumors while found quite frequently in the appendix are rarely seen in Meckel's diverticulum. Less than ten cases of carcinoid tumors have been reported in Meckel's diverticulum. In our series, excluding those containing aberrant gastric mucosa, only one benign tumor was found.

Malignant tumors are rarely found in Meckel's diverticulum. In the carcinomatous group Costich and McNamara (1946) cite previously reported cases and add an additional case. Of these cases seven were adenocarcinomas and two were of the medullary type. In the sarcomatous group Nygaard and Walters found fourteen cases.

As mentioned previously the preoperative diagnosis of Meckel's diverticulum is rarely made and, if made, it is impossible to state that a tumor is present. The pathologic complications such as inflammation, obstruction, perforation, hemorrhage, intussusception, etc., occur in non-malignant lesions as well as malignant lesions and, of course, much more frequently.

No malignant lesions were found in our series.

CASE XI. No. 93044, a girl, aged twenty, was admitted to the hospital complaining of pain in the right abdomen, nausea and vomiting of twelve hours' duration. The pain began the previous morning. It was located in the right lower quadrant and was intermittent in character. The patient was nauseated and vomited several times. On admission the patient stated that the pain was steady and in the right lower quadrant. The past history revealed that she had seven similar episodes during the past year.

Physical examination revealed a well developed, well nourished white female who did not appear acutely ill. Her temperature was 99.2°F., pulse 80 and respirations 18. The blood pressure was 120/80 and the red and white blood counts were normal. The urinalysis also was normal. The head, neck, heart and lungs showed no unusual findings. The abdomen was flat, soft, without palpable masses and was slightly tender over McBurney's point. A pelvic and rectal examination was negative. A preoperative diagnosis of subsiding appendicitis was made. Because of her numerous previous episodes an appendectomy was advised.

The abdomen was explored through a right pararectus incision. The appendix was slightly inflamed and was removed. Exploration of the ileum revealed an occluded Meckel's diverticulum 2 by 4 cm. about 12 inches from the ileocecal valve. Two and one-half inches of the ileum were resected with the diverticulum intact and a side-to-side anastomosis was performed. Exploration of other organs revealed no additional disorder and the abdomen was closed in layers. The patient had an uneventful recovery and was discharged on the twelfth postoperative day.

The pathologic report revealed that the diverticulum was filled with an amorphous, inspissated material. At the mouth of the diverticulum the mucosa was entirely absent being replaced by tightly packed caseous material surrounded by chronic inflammatory changes. (Figs. 7 and 8.) In the distal two-thirds of the diverticulum the caseous material had dissected between the muscularis and circular muscular coats producing detachment and eversion of the mucosal lining of the diverticulum. The diagnosis was an enterogenous cyst within a Meckel's diverticulum.

Enterogenous cysts anywhere along the outer

gastrointestinal tract are very rare. A search of the literature does not reveal any of such cysts contained in a Meckel's diverticulum.

ANALYSIS

Thirty-nine patients with Meckel's diverticulum were operated upon by various members of the surgical staff of the Jackson Clinic during the years 1915 to 1948. The preoperative diagnosis was correct in four of the five cases showing intestinal hemorrhage. The earliest case (1915) was not diagnosed preoperatively.

Meckel's diverticulum occurred twice as often in males as in females (twenty-six and thirteen). The average age, excluding the youngest, aged ten months, and the oldest, aged seventy-two, was twenty-five years.

Forty-six per cent (seventeen cases) of the diverticula were without disease. Fifty-three per cent (twenty-two cases) showed pathologic changes. Contrary to what has previously appeared in the literature, our inflammatory group (eleven cases) was the most dominant rather than the obstructive group. Of seven of the acute or gangrenous cases three went on to perforation with resulting peritonitis. There were four obstructive cases. In the peptic ulcer group (six cases) five showed clinical evidence of intestinal hemorrhage.

Abdominal pain was present in every case except the diastasis recti case in which the diverticulum was an incidental finding. Intestinal hemorrhage or melena was found in six cases. Fourteen patients vomited one or more times.

Resection or excision of the Meckel's diverticulum was performed in thirty-five of the thirty-nine cases.

All the patients went on to recovery except the very youngest and the very oldest. The former had a gangrenous ileum and expired on the first postoperative day (1933). The latter had generalized peritonitis due to a perforated diverticulum and expired on the nineteenth postoperative day (1926).

December, 1949

SUMMARY

1. Some historical aspects are discussed.
2. The pathologic complications of Meckel's diverticula are reviewed.
3. An analysis of thirty-nine cases and a detailed description of eleven cases illustrating the various pathologic conditions are presented.
4. A search for Meckel's diverticulum should always be made during an intra-abdominal operation, especially when the gross appearance of the organ being investigated does not fit the clinical picture.

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DEVELOPMENT OF TOTAL PNEUMONECTOMY*

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TOTAL pneumonectomy as first performed seventeen years ago was far from exhibiting a work of fine or polished technic. If any merit attaches, it rests on the daring nature of the procedure; however, the requisite courage reflected more credit upon the patient than upon the surgeon.

After reporting this case I did not make it the object of further publications and, consequently, no follow-up report was published. Now the opportunity is presented to make up for this omission. However, it must be understood that as early as two years after the first pneumonectomy the technic performed in this case had become obsolete.

At any rate it may be interesting to students of the recent history of medicine to appreciate the surgeon's fears and doubts before such an extensive operation especially since the only three recorded previous attempts had been unsuccessful.

When I became Sauerbruch's associate in 1921 the situation was somewhat as follows: The great hopes arising from the development of methods to obviate the danger of an operative pneumothorax were not realized in respect of pulmonary resection. We soon learned that a successful outcome on these operations was due to an obliterated pleura which made reopening of the ligated bronchus more or less harmless. In fact, closure of the bronchus was the paramount problem and it was thought that the wisest policy might be to anticipate a separation of the bronchial stump.

It became a fixed rule that obliteration of the pleural leaves must precede a successful lobectomy and much inventiveness was employed to produce this adhesive pleurisy. The hilum of the lobe was closed by placing an elastic tourniquet around the hilum or by mass-constricting ligatures waiting for the devascularized tissue to sequestrate. Thereupon the open lumen of the bronchus emptied its contents into a walled-off operative field.

The results of this cumbersome procedure were not too bad, the postoperative mortality of lobectomies being between 10 and 15 per cent.

In 1929 Harold Brunn published his paper on one-stage lobectomy in a free pleural space. From the practical and historical angle this represented considerable progress as was immediately realized in the Anglo-Saxon countries. However, the amazing results obtained by Brunn were not so much due to the particular handling of the hilum as to the institution of an intercostal subaqueous drainage in anticipation of a possible bronchial fistula. Thus removal of air and exudates and expansion of the remaining lobe were assured.

When the problem arose of removing an entire lung, we thought it was desirable to stick to the old method because of the accumulated experiences with this procedure in lobectomies. The problem, however, was not so much that of technic but of functional pathology. There was little concern as to the sudden loss of respiratory surface effected by pneumonectomy since traumatic surgery and collapsotherapy had amply shown how well defunctionalizing of an entire lung is tolerated. There was more concern about the empty space left by the removal of an entire lung. To be sure, several cases of agenesis of one lung were

* Read at the Historical Section of the American Medical Association meeting, 1948, at Chicago, Ill., on invitation by the Secretary who chose the title "The First Successful Pneumonectomy and its Historical Significance."

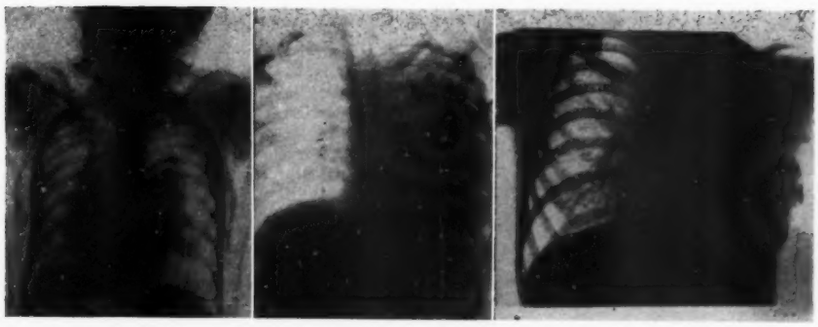

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FIG. 1. X-ray picture taken after admission of the patient on whom the first successful total pneumonectomy was performed (Surgical University Hospital of the Charite, Berlin). Pressure pneumothorax on the left side with displacement of the mediastinum; mediastinal and subcutaneous emphysema.
FIG. 2. June, 1931, after the patient had recovered from the immediate consequences of the accident, a bronchogram was performed which revealed bronchiectasis of the entire left lung.
FIG. 3. X-ray picture taken six months after left total pneumonectomy (July 21, 1931); retraction of mediastinum toward the side operated upon, extreme elevation of the diaphragm and shrinking of the left thoracic cage.

reported in the literature and the authors had clearly explained the way in which by shifting of mediastinum, elevation of diaphragm and shrinkage of the corresponding thoracic cage this problem was solved. The greatest apprehension, however, centered around the sudden occlusion of the main branch of the pulmonary artery. The disastrous effect of pulmonary embolism was all too familiar to us and we knew that shutting off one of the main branches meant a high mortality.

A review of the literature on experimental pneumonectomy disclosed that the operation had been successfully performed on rabbits and dogs in many instances even as far back as 1881 and 1882 by Gluck and by Biondi. In these experiments the question of adequate bronchial closure and of provision for compensatory filling of the empty half of the chest by the remaining thoracic organs is widely discussed, particularly in papers of the later investigators, but the circulatory problem arising from the ligation of a main artery and the two veins is scarcely mentioned. This is justified, I think, because the common experimental animals do not respond to massive pulmonary embolism in the way human beings do probably because of the tremendous reserve power of their hearts.

CASE REPORT

The opportunity of attempting pneumonectomy presented itself to me in July, 1931. The patient, a girl twelve years of age, developed severe mediastinal emphysema following a crushing injury to the thorax. An immediate incision in the neck resulted in liberation of air through the incision. The simultaneous presence of tension pneumothorax suggested a tear in the left main bronchus. (Fig. 1.) Empyema of the left pleura developed and drainage was instituted.

During the course of several months the typical symptoms of chronic pulmonary suppuration developed. An abscess which presented in the third (left) intercostal space near the sternum was drained. The wound communicated with the left main bronchus. Lipiodol injected through the fistula demonstrated bronchiectasis of the entire left lung. (Fig. 2.) Bronchostenosis resulting from the injury to the main bronchus was the obvious cause of the pulmonary suppuration. Although the patient's general condition was not good, it was decided to extirpate the entire left lung.

The two possible operative procedures were either total pneumonectomy or multiple unilateral lobectomy. In favor of the latter was our comparatively large experience with lobectomy. However, in performing lower and upper lobectomy a considerable portion of infected and dilated bronchi at the hilum would have to be left behind because of the low level of

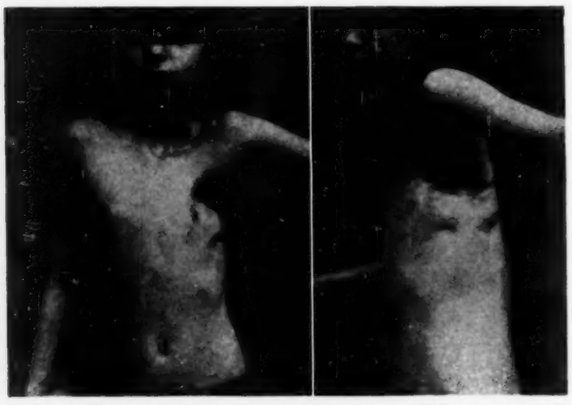

FIG. 4. Picture of the patient taken in March, 1932; the left axillary incision leading to a small fistula of the left main bronchus is visible. This fistula closed spontaneously shortly afterward.

dissection. Therefore, total pneumonectomy seemed to be the logical procedure with the understanding that the tourniquet should be placed as high on the hilum as possible.

After paralyzing the left diaphragm an axillary resection of the third to fifth rib was performed. The extensive adhesions offered no difficulty in separation of the lung. After the pedicle of the lower lobe had been developed and while endeavoring to isolate the upper lobe, strong traction on the hilum resulted in temporary cardiac stoppage. The operation was discontinued and the entire wound was packed.

I believe it was fortunate that the cardiac arrest occurred before the tourniquet was placed because otherwise we would have attributed it to occlusion of the major blood vessels rather than to the actual cause, a vagal reflex.

The second stage was performed fourteen days later on July 21, 1931. Beginning peripherally, the upper lobe was freed. After the entire lung was separated, an elastic tube ligature was tied around the hilum proximal to the bronchial obstruction; and additional silk ligatures were placed peripheral to the elastic ligature. Gauze was again packed around the lung.

After operation the patient's temperature was elevated to 39°C. and the pulse rate varied between 140 and 160 for about a week. The necrotic lung sloughed away on the fourteenth day. The residual cavity decreased rapidly in size, being aided by the extreme rise of the diaphragm and the marked retraction of the mediastinum toward the side operated upon. (Fig. 3.) Accordingly, a thoracoplastic collapse to obliterate the pleural space was not necessary. After eight weeks only a small fistulous tract which led to the stump of the left main bronchus remained.

The patient was demonstrated on March 31, 1932, at the annual Convention of the German Surgical Society (Fig. 4) and by Sauerbruch in April, 1934, before the same forum; the bronchial fistula had meanwhile closed spontaneously. The last picture of the patient was taken six years later in 1937. Her physical condition was excellent at this time; the left half of the chest showed signs of atrophy which was particularly evident in the different size of the breasts. (Fig. 5.)

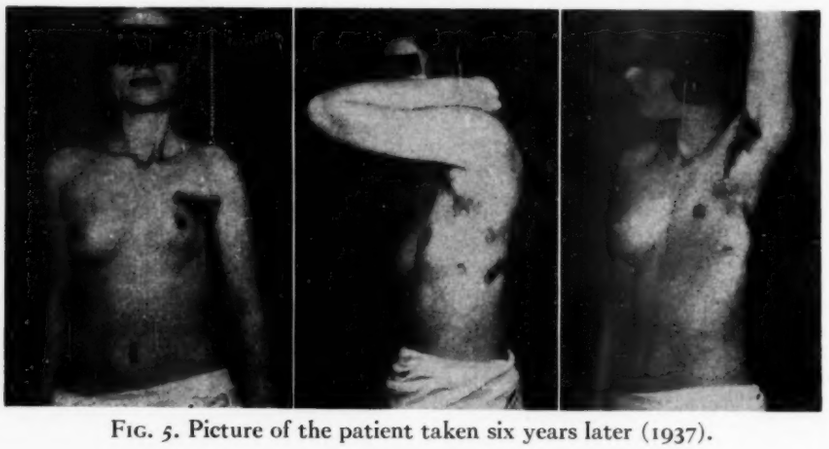

FIG. 5. Picture of the patient taken six years later (1937).

After the conclusion of hostilities I was unable to obtain news from the patient who is said to live in the Russian Zone of Occupation in Germany.

The outstanding feature during the second operation was the absence of acute symptoms following mass ligation of the

hilum vessels. Not even signs of vagal reflex, which forced us to discontinue the first attempt, were present. In spite of the tachycardia which persisted for almost a week, there was never noticeable dyspnea; this is in marked contrast to what is experienced in pulmonary embolism.

Another observation in our case was later shown to be the rule rather than the exception, namely, the fact that in spite of a limited rib resection, the empty half of the chest was filled with the remaining thoracic organs.

One and a half years after this operation the second successful pneumonectomy was performed by Cameron Haight on a girl of thirteen suffering from diffuse bronchiectasis of the left lung. The technic used by Haight was essentially the same except that the level of mass ligation chosen by him was different. He thought it wiser to ligate the two lobes separately leaving more bronchial tissue behind. While in this case a similar stenosis of the main bronchus was present as in my own, no unfavorable effects were observed from the presence of the two remaining stumps. Haight's patient is the oldest living pneumonectomy subject in this country. She is now married, has one child, and is in excellent general condition. She has had no pulmonary symptoms since her discharge from the hospital sixteen years ago. (Fig. 6.)

Fig. 6. Picture of Cameron Haight's patient twelve years after the left total pneumonectomy which was performed in 1932. (With the permission of Dr. Cameron Haight.)

The third successful operation, that of Evarts A. Graham, performed in April, 1933, has rightfully received much publicity because this time the pneumonectomy was done in one stage for carcinoma. When Haight and I ventured to remove a whole lung, it was our hope that surgical treatment of bronchogenic carcinoma might one day benefit from these experiences. The hope was quickly realized by Graham's brilliant operation and its splendid success. Graham's patient, a physician forty-eight years of age, today, fifteen years after the operation, is practicing and in the best of health. (Fig. 7.)

There was, however, one shortcoming in Graham's one-stage procedure. While it was fully successful in his particular case, it must be assumed that by leaving behind a large intrapleural stump the majority of carcinomas of the main bronchus could not be dealt with adequately by this technic.

The turning point in the technical development of total pneumonectomy, particularly for bronchogenic carcinoma, is represented by Rienhoff's successful mediastinal dissection.

Individual ligation of the three main vessels and suture of the main bronchus as first done by him was in accordance with surgical principles established for other organs of the body. Rienhoff's operation, successfully performed in July, 1933, for fibrosarcoma in a child three and one-half

FIG. 7. Picture of the patient on whom Evarts A. Graham performed total pneumonectomy for bronchogenic carcinoma in 1933. (GRAHAM, E. A., SINGER, J. J. and BALLON, H. C. Surgical Diseases of the Chest. P. 843. Philadelphia, 1935. Lea & Febiger.)

years of age, concluded the experimental stage of total pneumonectomy.

In the fifteen years which have passed since then very little has been added to this technic which beside achieving perfect hemostasis permits fairly reliable closure of the bronchus, which none of the previous technics had achieved.

Regarding the significance of the first successful pneumonectomy, this operation may have encouraged other surgeons by demonstrating first, sudden and permanent interruption of half of the vascular pulmonary circulation in human beings is, as a rule, well tolerated; second, the empty space remaining after pneumonectomy is rapidly filled out by compensatory expansion of the other lung and by displacement of heart and diaphragm.

However, the great practical value the operation has attained today must be accredited to Rienhoff and I believe that the operation which I performed seventeen years ago would never have justly been recorded in the textbooks of the history of medicine had not Rienhoff modified the operative technic.

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AN INQUIRY INTO LATE POSTPARTUM HEMORRHAGE*

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DESPITE the fact that late hemorrhage in the puerperium constitutes one of the most grave complications the obstetrician encounters and despite the fact that it requires keen diagnostic acumen and seasoned clinical judgment for its proper evaluation and correct management, there is remarkably little written on the subject. Other names for this complication are secondary puerperal hemorrhage, spätblutung and hemorrhage tardive. Isolated articles, consisting mostly of case reports, may be found in the French and German literature under the latter two titles. In contrast to the voluminous literature which was accumulated on the subject of immediate postpartum hemorrhage, aside from sporadic reports, there is practically nothing to be found regarding late puerperal hemorrhage, a complication just as serious if not even more treacherous than immediate postpartum hemorrhage.

The definition of late postpartum hemorrhage is entirely arbitrary. Kermauner[1] defined it as bleeding coming on twenty-four hours postpartum or postabortum, whereas Frankl[2] required that the bleeding not appear until at least a week after parturition or abortion to be considered under spätblutung. It has been my practice to regard the puerperium as lasting three months rather than just six weeks inasmuch as many patients have not completed the process of involution for approximately three months. Accordingly, the term late postpartum hemorrhage will be defined as any genital tract bleeding coming on after the first twenty-four hours of the puerperium postpartum or postabortum.

The seriousness of late postpartum hemorrhage is generally not appreciated. Secondary hemorrhages are often sudden and massive and may be as exsanguinating and shocking as hemorrhage from placenta previa, placental ablation or severe atony immediately postpartum. On the contrary, each episode of late bleeding may be only moderate in amount but the bouts may recur frequently or the bleeding may be an almost continuous issue of blood which may go on for weeks although the amount lost on any one day may not be excessive. Secondary hemorrhage is particularly treacherous inasmuch as it often happens days or weeks after the patient has been dismissed from the hospital hence, the armamentarium for combating shock and hemorrhage is usually not immediately available in contrast to the facilities customarily in readiness in all well equipped hospitals for the treatment of immediate postpartum hemorrhage. It is usually very difficult for the attendant to explain an episode of late puerperal hemorrhage to the patient and her family; sometimes it is quite difficult for him to satisfactorily explain it to himself. In any event the usual reaction of the patient is the belief that the attendant is guilty of a sin of omission. On the contrary, however, careful inquiry into the causation of the majority of these instances of late bleeding establishes the fact that the complication was entirely unforeseeable and unavoidable.

The frequency of late bleeding is difficult to estimate. However, no less than 10 per cent of puerperal patients experience excessive bleeding after the first twenty-four hours. Usually, to be sure, the bleeding in the average case responds to a prescription of limited activities, more bed rest and a course of oxytocics. Nevertheless, in my

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experience somewhat more than 2 per cent of my puerperal patients have required additional active treatment for late bleeding. Therapeutic measures have included blood transfusion, curettage, tamponade, vaginal ligation of the uterine arteries, hysterectomy and ovarian cystectomy. From a wide obstetrical experience it has been Stephenson's[3] observation that the incidence of late postpartum hemorrhage has definitely increased in the last five to ten years despite the fact that he has not employed estrogens for the prevention or suppression of lactation and despite the fact that the percentage of mothers nursing their babies has not changed. One wonders whether or not the practice of earlier ambulation and the vogue of patients resuming full activities so soon after return home following delivery may be contributory factors in the definitely increased incidence of secondary bleeding.

Investigation of patients who bleed excessively in the puerperium usually discloses some local pelvic disorder as the etiologic factor; most commonly, retained fetal elements, evidence of faulty regeneration of the uterine mucous membrane or submucous myomas are found. Kermauner[1] classified his cases into four groups as follows: (1) retained placental tissue; (2) generalized infections; (3) aneurysms and (4) defective regression of the blood vessels in the uterine mucous membrane. However, before proceeding with an inquiry into the specific pelvic lesions which can cause late hemorrhage, it might be of interest to review the general systemic disorders which may evidence late postpartum hemorrhage as a salient clinical manifestation.

HEMORRHAGE DUE TO SYSTEMIC DISORDERS

The Hypotonic Somatotype. In his very provocative book, "The Autonomic Diseases," Rivers[4] devotes three most interesting pages to a discussion of the difference between the hypertonic, with predominant thoracolumbar autonomic system, and the hypotonic (vagotonic) obstetrical patient. In speaking of the hypotonic type he says, "Some of this type hardly cease to menstruate during pregnancy, and they may have hemorrhages from the most trivial causes. Abortions are more frequent, and delivery sometimes results without pain; but the hemorrhages following delivery may be profuse and hard to control. They are not only susceptible to hemorrhage, but they are susceptible to shock, and every precaution must be observed to prevent unusual excitement which could cause shock, lest it bring sudden dilatation of the uterus and the vessels therein to result in fatal hemorrhage. Astringents have less pressor action on the uterus of this type and they need to be repeated more often. . . . While the tonic type requires special care to prevent toxemia, the hypotonic class requires special care to prevent hemorrhage."

I agree with Rivers that there is a hypotonic type of woman who is basically frail. Her blood pressure is usually subnormal or normal; she aborts easily; her labor is characterized by easy dilatation of the cervix; but she tends to have immediate postpartum hemorrhage from uterine atony and she seems prone to secondary hemorrhage during the puerperium. Whether all this is due to the vagotonia or whether the latter is simply part of the physiologic make-up of this constitutional type is not clear. In any event the hypotonic somatotype bleeds easily and sinks into irreversible shock from a blood loss usually not incompatible with life for certain other constitutional types.

Blood Dyscrasias. Secondary puerperal hemorrhage may be a manifestation of one of the hemorrhagic purpuras, aplastic anemia or leukemia. Thrombopenic purpura may be a disease *sui generis* or it may be secondary to infectious diseases (scarlet fever, tuberculosis, sepsis) or to the action of certain toxic or allergenic agents. Secondary purpura has followed the taking of iodine, bismuth, benzol and arsphenamine. Peshkin and Miller[5] have reported

the occurrence of purpura after the ingestion of ergot and quinine. Radiation therapy may induce hemorrhagic purpura by its depressant action upon the bone marrow just as metastatic tumor can cause it by myelophthisis. Rushmore[6] has reported thrombopenic purpura accompanying pregnancy and Minot[7] has described its association with menstruation. Menorrhagia may be the first and often the only symptom of thrombopenic purpura.

To cases of severe puerperal sepsis complicated by aplastic anemia Couvelaire[8] gave the name "metrorrhagic form of puerperal sepsis." Williams[9] reported such a case of desperate postpartum sepsis which ended fatally with hemorrhage late in the puerperium due to aplastic anemia. A fatal uterine hemorrhage on the eighth postpartum day was the termination of Mann's[10] case of pregnancy complicated by lymphatic leukemia.

From the aforementioned it is evident that one should always consider the possibility of blood dyscrasia in evaluating instances of secondary puerperal hemorrhage. A complete count, including platelets, bleeding and clotting time and a study of clot retraction, may be very revealing in late hemorrhage cases.

Capillary Fragility. Increased capillary fragility has been reported in a wide variety of conditions. According to Rodriguez and Root[11] capillary permeability is increased in hypertensive states, diabetes and tuberculosis and may be improved by the administration of rutin, the flavone glucoside. Kushlan[12] has observed a striking improvement in the bleeding tendency of patients with hereditary hemorrhagic telangiectasia after rutin therapy. The tendency for the chronically hypertensive puerpera to experience late hemorrhage may be related to abnormal capillary permeability known to accompany hypertensive states. In the days before rutin therapy Strauss[13] reported a desperate case of repeated hemorrhages in the puerperium due to what he called constitutional capillary fragility. The vaginal mucosa in his case required resuturing on five occasions and eventually the internal iliac arteries had to be ligated to control the persistent vaginal bleeding.

Vitamin Deficiency. Associated with abnormal bleeding tendencies is a deficiency of any one of four vitamins, namely, vitamin C, vitamin K, vitamin P (citrin) and vitamin B complex. In the scorbutic state there are large subcutaneous hemorrhages as well as bleeding from various body cavities. Vitamin P deficiency is manifest by universal capillary fragility which may be verified by the Göthlin petechial index. Inasmuch as vitamin K deficiency can allow bleeding from an extensive raw surface without bleeding from intact skin and mucosa, puerperal bleeding from the denuded uterine wall may be the only manifestation of vitamin K deficiency. The Biskinds[14] have written extensively upon the possible role of B complex deficiency, particularly thiamin and riboflavin, in the causation of menorrhagia. The theory is that the liver cannot conjugate and efficiently dispose of estrogens in the absence of adequate B vitamins, hence, the abnormal bleeding is thought to result from hyperestrinism. Therefore, it might be concluded that the obstetrical patient facing confinement with depleted vitamin stores is particularly liable to abnormal bleeding in the puerperium.

Liver Disease. The hemorrhagic tendency in jaundiced patients is due to the reduced blood level of prothrombin, which in turn is due to the decreased absorption of vitamin K, the antihemorrhagic vitamin. For the absorption of the latter it is necessary that bile be present in the intestine. In addition to the prothrombin deficiency women with liver disease probably develop hyperestrinism, as previously indicated. It will be recalled that the free hormone is considerably more potent than conjugated estrogen.

Recent studies[15] leave little doubt that acute yellow atrophy is an end stage of fulminant infectious hepatitis due to an icterogenic virus. Zondek and Bromberg[16]

encountered twenty-nine patients with infectious hepatitis among 3,382 pregnant women (0.85 per cent). Of the five fatal cases two women hemorrhaged shortly before death. I recall a patient who bled almost continuously for a week postpartum while in the preicteric phase of infectious hepatitis. Clinical jaundice did not appear until two days after curettage.

Although infectious hepatitis is the most frequent cause of icterus in pregnancy and the puerperium, it may result from hemolytic accidents (transfusion reactions) and from obstructive lesions of the biliary tract.

Hypothyroidism. The postpartum period seems to be one of the favorite phases in a woman's life for a previously normal or marginal thyroid to become hypoactive. One of the first manifestations of hypothyroidism may be menometrorrhagia. Thyroxine augments cellular activity throughout the body including, of course, the glands of internal secretion. According to the work of Van Horn[17] thyroxine tends to prevent the building up of excessive quantities of estrogenic hormone in the endometrium by promoting the elimination of estrogens through the urine. It is well known clinically that thyroid therapy is most effective in correcting instances of dysfunctional bleeding in hypothyroid women.

Heparin and Dicoumarin. *A priori* one would justifiably be wary of the use of anticoagulants such as heparin and dicoumarin in the prophylaxis and treatment of phlebothrombosis and thrombophlebitis because of the not improbable hazard of secondary postpartum hemorrhage. A more extended clinical experience with these anticoagulants will be necessary before the final answer is forthcoming.

HEMORRHAGE DUE TO GYNECIC DISORDERS

By far the great majority of instances of late postpartum hemorrhage arises in connection with a specific gynecic condition most commonly from a pathologic state of the uterine body but occasionally from a lesion in the vulvovaginal tract, the uterine cervix or the adnexa. It may be of interest, therefore, to review the gamut of pathologic conditions arising within the reproductive system which may be associated with late puerperal bleeding.

The Vulvovaginal Tract

Ulcerative lesions of the vaginal tract, vaginal metastases from choriocarcinoma and rupture of a vaginal varix may cause bleeding late in the puerperium but perhaps the most frequent causes of delayed bleeding from the vulvovaginal tract are as follows:

Vulvovaginal Hematoma. This may follow spontaneous as well as operative vaginal delivery and it is often quite insidious in onset. According to Duckman and Tortora[18] only 187 cases have been reported since the first recorded instance in 1554; however, the condition is doubtless much more common than the number of reported cases would indicate. A paravaginal hematoma may arise in association with episiotomy or without incision of the perineum. Perineal pain, arising a day or two postpartum and which is unusually severe and makes little or no response to opiates, should suggest the possibility of a developing vulvovaginal hematoma. One recent case, attended by myself, manifested itself on the tenth postpartum day following delivery by outlet forceps and mediolateral episiotomy on the side opposite to which the hematoma developed. Approximately 500 cc. of liquid and clotted blood escaped from the hematoma several days after the patient's return home from the hospital. Readmission was necessary for tamponade and administration of blood transfusion. It was believed that the hematoma formed as a result of the spontaneous rupture of a vulvar varicosity of which the patient, although a primipara, had many. Large hematomas, which do not resorb or which are not spontaneously or surgically drained, have been known to dissect along the loose cellular tissues to eventuate in a broad ligament or retroperitoneal hematoma. Although perineal

pain may suggest the possibility of a hematoma developing, it may be a week or more before the tumorous collection of blood is evident. With the present tendency for brief hospital stay postpartum, the attendant may not become aware of a vulvovaginal hematoma until spontaneous rupture or secondary infection of the lesion has occurred. With hematomas which develop in connection with episiotomy, however, the tumor is usually obvious within twelve to twenty-four hours postpartum.

Rupture of the Vaginal Vault during Coitus. Although a rare accident, according to Diddle,[19] 133 major tears of the vaginal vault resulting from coitus have been recorded in the past 76 years. This figure includes the two recent cases encountered by Diddle. Of the 133 cases, 22 patients died giving a mortality of 16.7 per cent for the accident. The usual symptoms are vaginal pain during coition with bleeding thereafter. Diddle's first patient was admitted because of moderately severe vaginal bleeding coming on after first coition following delivery two months previously. Examination disclosed a crescentic tear 4 cm. long involving the entire thickness of the vaginal wall. His second case occurred in a colored primipara who returned to the hospital sixteen days after normal parturition. The patient admitted having had coitus three days before return to the hospital. Examination disclosed a gaping tear in the left posterior vaginal fornix. Treatment consisted of blood transfusion, rest and hot vaginal douches. Healing was nearly complete within a week.

Although rupture of the vaginal vault during coitus is an exceedingly rare lesion in private obstetrical practice, it is sometimes encountered in clinic patients. The importance of a full history and careful speculum examination should not be overlooked.

Vaginal Wall Lacerations. Secondary infection in a repaired vaginal wall laceration may evoke premature digestion of the suture material with sudden brisk bleeding in the early puerperium. Even with careful hemostasis a hematoma may form under the suture line of a vaginal laceration, become infected and rupture usually five to eight days postpartum.

Vaginal Cuff Following Complete Hysterectomy. Several years ago at the Maternity Hospital of Cleveland I had the opportunity of attending a patient who had undergone total hysterectomy several hours postpartum because of rupture of a myomatous uterus which followed breech extraction. Sudden massive, almost exsanguinating postoperative hemorrhage occurred about three weeks postpartum and was found to be due to the erosion of an angular artery in the vaginal cuff. The patient recovered following transfixion of the bleeding artery, vaginal tamponade and generous blood replacement.

Cesarean section followed by immediate total hysterectomy and total hysterectomy done soon postpartum (for severe atony or rupture) are uncommonly performed operations. Also, the circumstances under which they are done are typically characterized by expediency and haste; hence the usual precautions in transfixing the vaginal cuff vessels may not be observed and, moreover, the increased vascularity of the parts in the pregnant state renders good hemostasis difficult to accomplish. The risk of serious late bleeding in these cases is a real hazard, particularly inasmuch as it may not happen until several days after the patient has returned home as occurred in the case previously cited.

The Uterine Cervix

Although the uterine cervix itself is a rare source of secondary puerperal hemorrhage, there are several pathologic entities which should be considered in the differential diagnosis of delayed bleeding.

Lacerations. Repaired lacerations of the uterine cervix may give rise to late bleeding in one of several ways: Infection along the suture line may cause premature digestion of the catgut with late hemorrhage on about the seventh day. The descending

cervical branch of the uterine artery may hemorrhage in the early puerperium if the primary suturing of the upper extremity of the laceration was not carefully performed; proper closure of the upper angle of extensive cervical tears is the most essential step in the primary repair as far as permanent hemostasis is concerned. When one considers how the azygos vaginal artery anastomoses with the descending cervical rami of the uterine arteries to form an arterial circle, called the coronary artery of the cervix, it is remarkable that there are not more instances of secondary hemorrhage from aneurysm formation in connection with lacerations which involve major arterial channels. What has been said previously regarding lacerations of the cervix applies equally as much to trachelotomy or Dührssen's incisions of the uterine cervix. Extensive cervical lacerations which are not immediately recognized and repaired are properly considered under primary postpartum hemorrhage as the bleeding continues postpartum until repair is made or the patient exsanguinates herself.

Carcinoma. A hitherto unrecognized cervical carcinoma may ulcerate and bleed in the puerperium and elicit secondary hemorrhage. I can recall such an occurrence in an unregistered patient who first hemorrhaged on the seventh postpartum day. In a recent study of the postpartum cervix Sheets[20] encountered two cases of clinically unsuspected, grossly not discernible, invasive carcinoma of the cervix in an unselected series of 200 postpartum cervices. From a histologic study of 1,200 specimens removed in the course of panhysterectomy Pund and Auerbach[21] diagnosed unsuspected carcinoma in 3.9 per cent of the cervices. Hence the figures usually cited for the incidence of carcinoma of the cervix coincident with pregnancy (one case in 2,000 to 3,000 deliveries) may have to be revised. A contemplation of these two studies previously cited would behoove the attendant to consider the possibility of cervical carcinoma in the differential diagnosis of secondary puerperal hemorrhage particularly in the older multiparous parturient.

Cervical Stump Following Supravaginal Hysterectomy. Even after cesarean-supravaginal hysterectomy there is a risk of late hemorrhage. In his series of 168 cases of cesarean-hysterectomy Dieckmann[22] encountered among the postoperative complications two instances of hemorrhage from the cervical stump on the tenth postpartum day. Both cases were managed by the application of clamps to the cervix for forty-eight hours; neither patient required a blood transfusion. With the wider vogue which cesarean-hysterectomy is currently enjoying, one may encounter more instances of secondary hemorrhage from the remaining stump.

Miscellany. Cervical erosion, cervical polypi and papillary cervicitis may cause bleeding in the puerperium. Although cervical varicosities are rare and although they usually bleed during pregnancy if they are going to bleed, they should be remembered as possible sources of delayed bleeding in the puerperium. The importance of thorough visualization of the cervix and vaginal tract in instances of late bleeding cannot be overemphasized.

The Corpus Uteri

The overwhelming majority of instances of secondary puerperal hemorrhage arises from pathologic conditions of the uterine body. A wide range of disease entities is represented in a scrutiny of the pathogenesis of late postpartum hemorrhage from this source. The various pathologic entities will be reviewed in the order of their clinical frequency. However, before proceeding with the specific entities it might be appropriate to review briefly the physiology and management of the third stage of labor which is exceedingly important in the prevention of both primary and secondary postpartum hemorrhage.

The proper conduct of the third stage of labor demands first that the baby be delivered slowly; Dieckmann[23] recommends that three minutes be so spent. This is to

give the uterus time to contract slowly about the placenta and thus detach it by a squeezing process. The retroplacental hematoma has little or nothing to do with separation of the placenta if the delivery of the child is conducted slowly and if the third stage of labor is properly managed. The Schultze and Duncan types of placental presentation have nothing to do with the process of placental separation and depend merely upon whether or not the detached placenta encounters a tight ring such as a contracted lower uterine segment or vaginal orifice on the way out of the birth canal.

No attempt should be made to deliver the placenta until the uterus has regained its globular form when the fundus should be gently compressed provided the uterus is definitely firm and not atonic. The fundus should never be pushed into the axis of the pelvis for risk of inversion. If the placenta lies free in the vagina, it should be delivered by gentle traction on the cord. The administration of pituitary extract after the second stage promotes contraction and retraction of the uterus and placental separation. Relaxation of the fundus with bleeding in the third stage calls for stimulation by massage, bimanual compression and repetition of an oxytocic agent. Should these measures fail, the placenta should be extracted manually. If uterine bleeding continues after the placenta has been delivered intact, the uterine cavity should be manually explored to exclude a retained succenturiate lobe, retained blighted ovum of a multiple pregnancy,[24] myoma, rupture and inversion. The observation that the placenta presents a punched-out defect on its maternal aspect should suggest the presence of a submucous myoma. I believe that in modern obstetric practice there is still an occasional call for the timely use and proper application of a uterine pack particularly in instances of severe atony. Such tampons, if employed, should rarely be allowed to remain *in situ* for more than eight hours. Once in a great while primary atony may even demand the timely use of hysterectomy. *Pari passu* with the local measures mentioned, blood replacement therapy must be prompt and adequate.

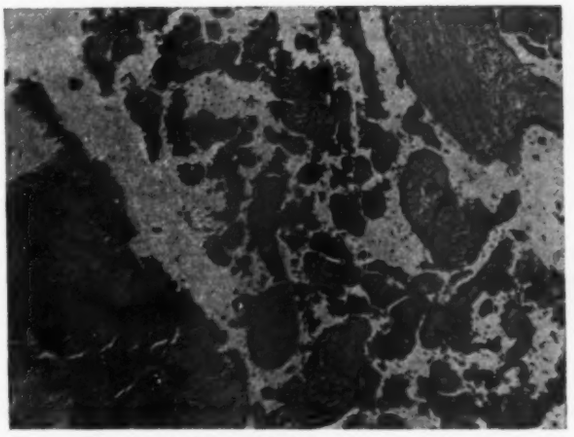

FIG. 1. Retained placental tissue removed by curettage on nineteenth postpartum day; nubbin of tissue was missed on curettage done on twelfth postpartum day when only evidence of chronic endometritis was found on microscopic examination.

Retained Placental Tissue. The most frequent cause of late postpartum hemorrhage is retention of placental tissue. The amount of placenta retained may vary from a small nubbin (Fig. 1) or lobule to an entire accessory lobe. Even the most careful scrutiny of the delivered placenta may fail to disclose the missing fragment of a cotyledon which later may become the source of serious delayed bleeding. With retained placental tissue there is typically no let-up of the lochia rubra, and brisk hemorrhage occurs when, during the process of involution, the uterus contracts around the retained tissue partially detaching it from the uterine wall. Partially detached tissue can hold open a blood vessel and interfere with retraction so that the vessel continues to bleed; not until the tissue is removed will the vessel retract and the bleeding cease. A fragment of placental tissue may gradually become organized and incorporated as an integral part of the uterine wall; such a placental polyp may be recovered weeks, months or even years later on curettage or hysterectomy for abnormal uterine bleeding. Dorsey[25] reports an instance of sudden

profuse hemorrhage (1,500 cc.) on the twenty-second postpartum day from a small placental polyp. Hagstrom[26] records an interesting instance of desperate late hemorrhage occurring in a primipara on the eighteenth postpartum day. Curettage provoked such brisk hemorrhage that hysterectomy had to be carried out as a life-saving procedure. The pathologic finding was a simple placental polyp. It should be emphasized that despite all due vigilance and care in the conduct of the third stage of labor and keen scrutiny of the afterbirth, occasional instances of retention of bits of placental tissue do occur. Almost always in such cases review of the original delivery record will describe the placenta as having been delivered whole and intact.

Succenturiate Lobe. According to Waters[27] succenturiate lobe occurs about once in 500 deliveries. Retention of such an accessory lobe may be a most treacherous source of delayed hemorrhage. When the placenta has been described as intact at the time of delivery but a copious amount of placental tissue is obtained on curettage performed a week or so later for secondary hemorrhage, a very substantial number of such instances is doubtless due to a missed succenturiate lobe. It is important to check the delivered placenta for gaping, sizeable marginal vessels which would indicate vascular connection with a retained succenturiate lobe. On the other hand, it rarely happens that the small accessory lobe is delivered first. If the placenta is inordinately small for the size of the fetus, it behooves the attendant to explore the uterine cavity forthwith for the main placenta. I once observed placenta previa to be due to a succenturiate lobe and Waters[27] reports a case of placenta previa due to a succenturiate lobe 10 cm. in diameter. What has been said concerning succenturiate lobe applies equally as much to the retention of half of a duplex placenta. Just recently I encountered at cesarean section the retained half of a duplex placenta, the first half having come away spontaneously and quite intact.

If a cotyledon of placenta, a succenturiate lobe or half of a duplex placenta is retained, the lochia rubra persists beyond the seventh day, there are bouts of brisk bleeding, the uterus remains boggy and subinvoluted and often there is a low-grade fever. According to Wolfe and Pedowitz[28] the cervix is normally patulous until the twelfth postpartum day; however, finding the cervix patulous after that time is highly suggestive of retained placental tissue. Bleeding will continue as long as the partially detached placental tissue keeps the underlying blood vessels open and unable to retract. The indication for prompt curettage as soon as the diagnosis is reasonably established is obvious.

Placenta Previa Accreta. Chisholm[29] recently reported a most interesting case of placenta previa accreta occurring in a thirty-five year old secundipara. The patient's first puerperium was complicated by secondary postpartum hemorrhage six weeks after delivery for which curettage was carried out. The second pregnancy was complicated by placenta previa for which cesarean section was carried out in the thirty-fourth week. At operation an accreta placenta previa was encountered and "all that safely could be done was to remove as much as possible of the placenta piecemeal." Late postpartum bleeding began on the sixteenth day and a firm pack was inserted into the cervical canal on the seventeenth day. By the nineteenth day of the puerperium the patient's condition had sufficiently improved so that total abdominal hysterectomy could be undertaken. The patient gradually improved and was discharged from the hospital on the fifty-sixth day. Microscopic examination of the uterus showed a total absence of the decidua basalis as well as a poorly defined Nitabuch's membrane so that the villi lay mostly on the muscular layer.

Retained blood clots or *membranes* alone are rarely the cause of late postpartum hemorrhage although occasionally careful evacuation of the puerperal uterus may fail to yield anything else. In such instances

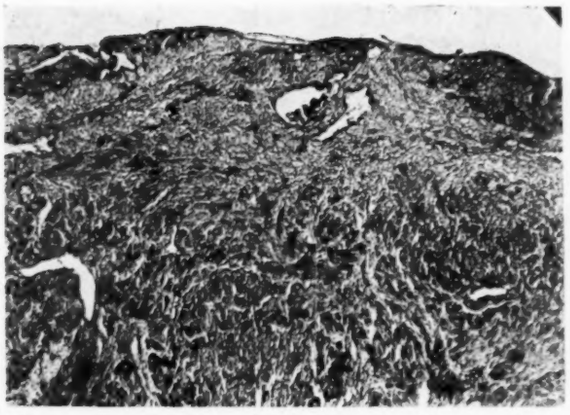

FIG. 2. Section through decidua basalis, cesarean-hysterectomy specimen, showing minimal spongy layer remaining and absence of distended blood vessels; the numerous fetal giant cells are diagnostic of the placental site. By forty-five minutes postpartum the blood vessels of the placental site become distended and partially thrombosed.

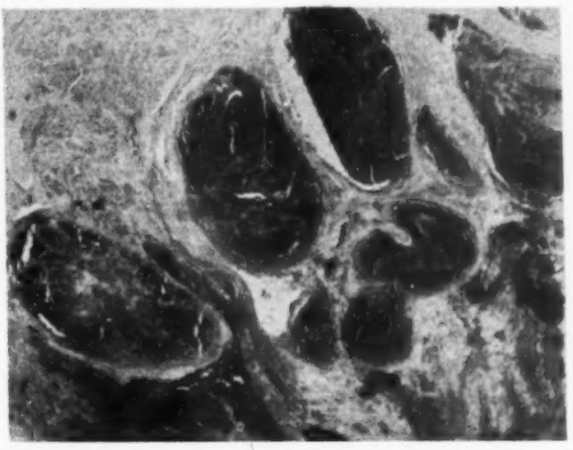

FIG. 3. Section taken through normal placental site thirty-two hours postpartum (cesarean section); patient expired from ruptured congenital aneurysm of circle of Willis one month from term. Section shows remarkable thrombotic obliteration of placental site veins.

one always wonders if perhaps a nubbin of placental tissue were not "missed" or some other pathologic entity not detected.

Persistent Placental Site. The anatomic changes which occur in the uterine mucosa postpartum or postabortum are indeed fascinating. A cleavage plane appears in the decidua vera by the second or third day postpartum, dividing the spongy layer into an inner necrotic zone and an outer, well-preserved lamella which adjoins the muscularis. The inner necrotic zone is separated by a process of ischemic necrosis due to vasospasm of the spiral arterioles; the process is indeed like menstruation. The desquamated layer is cast off in the lochia whereas the preserved inner lamella contains the fundi of the glands and constitutes the matrix whence the new endometrium is regenerated. Approximately seven to ten days are required for the new endometrium to cover completely the area previously occupied by the decidua vera.

The fate of the placental site, however, is a totally different process. In his last paper Williams[30] presented a scholarly exposition of the changes which take place in the placental site; similarly, Teacher[31] devoted much study to the healing of the placental site. It has been established that the placental site blood vessels, both arteries and veins, start their process of obliteration during the second stage of labor and continue it during and after completion of the third stage. (Fig. 2.) Vascular obliteration is accomplished by venous sinus thrombosis and obliterating endarteritis. (Fig. 3.) The placental site is not absorbed *in situ* but rather heals by a process of exfoliation in which the placental site is virtually "lifted off" by the growth of a new epithelium from the periphery as well as by the sprouting of regenerated mucosa from the gland stubs underlying the placental site. Williams[32] found an exfoliation of tissue from the placental site during the entire first six weeks postpartum; hence, about six weeks are usually required for disappearance of the placental site although with the unaided eye it is most difficult to detect the placental site by ten to fourteen days postpartum according to Rutherford.[33]

Occasionally there is delay in extrusion of the placental site. According to Rutherford[33] placental sites have been reported as late as nine weeks postpartum. Williams[30] maintained that both intra-uterine infection and the retention of fetal tissue definitely delayed involution of the placental site. The clinical picture of subinvolution of the placental site is practically identical with that of retained placental

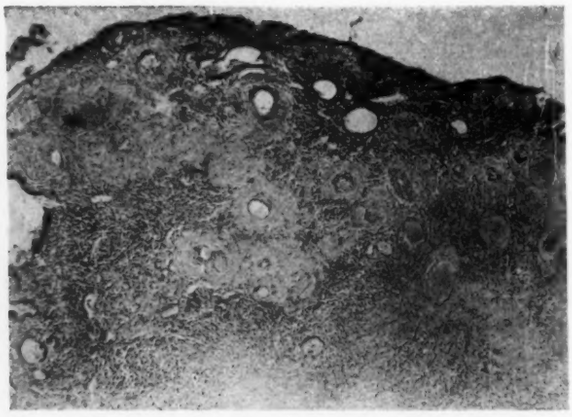

FIG. 4. Persistent placental site; specimen obtained in late puerperium from secondary postpartum hemorrhage case. Note patency of blood vessels. (Specimen courtesy of Herbert F. Traut, M.D.)

tissue: (1) lochia rubra persisting beyond the seventh day or bouts of frank uterine bleeding; (2) subinvolution of the uterus as a whole and (3) usually, low-grade pyrexia of 99 to 100°F. Faulty involution of the placental site is really a rather frequent cause of late bleeding following either abortion or term delivery. Erviny and Power[34] regarded it as quite common for in their series of 134 postpartum evacuations for late bleeding they recovered actual placental tissue in only ten instances. Rutherford and Hertig[35] made a careful study of three cases of serious secondary puerperal hemorrhage proved to be due to non-involution of the placental site. The characteristic finding on pathologic examination of the tissue removed was patent or incompletely obliterated blood vessels. (Fig. 4.) All three patients were curetted but the third one required hysterectomy eight days after curettage because of continued bleeding. These authors believe that the condition is more likely to occur in multiparas and that there is a tendency for it to recur in subsequent pregnancies. The mechanism of bleeding is obviously the result of hemorrhage from the placental site blood vessels which remain patent, or at least incompletely obliterated, at the time the placental site is in the process of being extruded. Dieckmann[23] encountered persistent placental site as the most frequent finding in the patients readmitted for curettage for late puerperal bleeding; only rarely was placental tissue obtained in his patients at Chicago Lying-In Hospital.

In conclusion it might be stated that persistence or non-involution of the placental site is a common cause of secondary postpartum hemorrhage. The reason for failure of proper vascular obliteration is not known. The bleeding may be so profuse or so prolonged that even hysterectomy may be mandatory. This phenomenon may follow abortion as well as term delivery. McSweeney[36] encountered it as the cause of death in one patient who expired on her twenty-third day following delivery by cesarean section.

Blighted Secondary Ovum. Leff[24] calls attention to the possibility of retention of a blighted secondary ovum of a multiple pregnancy as a hitherto overlooked cause of secondary postpartum hemorrhage. Leff believes that when there is an interlude of a number of days of normal lochia before actual bleeding begins, one can be quite sure he is not dealing with retained secundines. In one of Leff's cases curettage for late bleeding yielded a cast of an embryo which measured 5 by 3 cm. The smaller the retained ovum the longer it will be before the uterus involutes to a small enough size to encroach upon the retained ovum and cause its detachment. Bleeding results from partial separation of the retained tissue, with hemorrhage from the torn vessels which cannot retract as long as the tissue remains attached. Some of Leff's cases occurred as late as thirty days postpartum.

If the placenta as originally delivered is described as whole and intact and if one is sure there was no succenturiate lobe involved, the finding of placental tissue on curettage for late bleeding suggests, according to Leff, that one is dealing with the residue of a blighted secondary ovum of what started out as a multiple pregnancy.

Decidual Separation. In his classical paper Williams[37] pointed out that no hard and fast rules can be laid down as to just

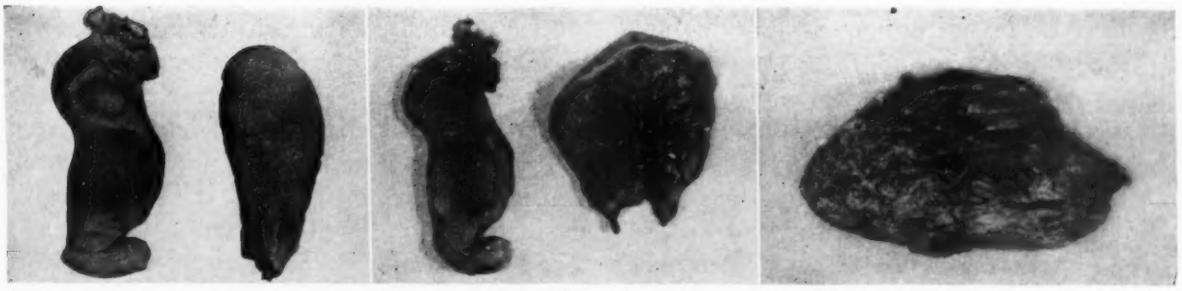

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FIG. 5. Tubal pregnancy (left) and decidual cast. (Specimen courtesy of Robert L. Faulkner, M.D.)
FIG. 6. Tubal pregnancy (left) and decidual cast bisected. (Courtesy of Robert L. Faulkner, M.D.)
FIG. 7. Decidual cast from case of tubal pregnancy; note extensive hemorrhage throughout cast. (Courtesy of Robert L. Faulkner, M.D.)

how much decidua will be retained either in the placental site or elsewhere in the postpartum uterus. He found, in actual fact, that the amount of decidua retained varied considerably in different specimens; there were all graduations from a thick layer on the one extreme to "minute decidual triangles between the serrated margins of the muscularis on the other." In 1926 Frankl[2] published an interesting study of late postpartum hemorrhage due to hyalinized vascularized decidual rests or islands. Of a series of 460 patients who first bled at least a week postpartum or postabortum, the author collected six hysterectomy specimens from patients who presented no other organic pelvic disease and who showed no histologic evidence of retained chorionic tissue. The only pathologic finding in this carefully selected series of cases was vascularized decidual rests. To the latter Küster[38] is credited with having given the name deciduoma. Frankl[2] hypothecated that the retained islands of decidua became unduly vascularized by what he called the plexus venosus varicosus endometrii. On gross scrutiny of the excised uteri the islands of vascularized decidua could not be differentiated from nubbins or plaques of retained placenta. In his textbook Williams[39] mentions how he himself had been "surprised to find that a portion of decidua only a few millimeters in diameter may lead to serious consequences" as far as puerperal bleeding is concerned.

Palik and Rechnitz[40] recently reported a series of nine cases of hemorrhage late in

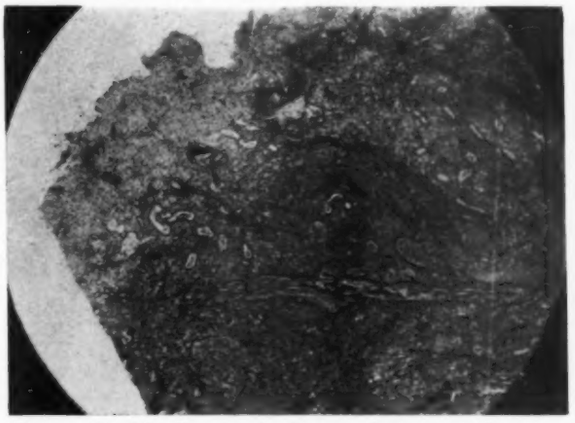

FIG. 8. Decidual cast; the hemangiomatous character of the normal cast is striking. When separation occurs, the bleeding may be profuse and alarming. × 20.

the puerperium, five after abortion and four after full-term delivery. In each instance the only cause of bleeding that could be ascertained on study of the curettings was hyaline degeneration of decidual rests.

In addition to the pathologic entity of hyalinized vascularized decidua previously described serious late bleeding may arise from the separation of really quite normal decidua. I have encountered this complication a week or more after salpingectomy for tubal pregnancy (Figs. 5, 6, and 7). Richly vascularized decidua (Fig. 8) was the only finding on pathologic examination of the uterine curettings. Were it not for the frequency with which curettage is done as a diagnostic procedure before laparotomy in so many cases of ectopic pregnancy, this complication of late bleeding would no doubt be more commonly en-

countered. The writer can recall an instance of late postpartum bleeding which arose from the non-pregnant horn of a double uterus in a patient who had been delivered over a week previously by cesarean section.

Subinvolution. Involution is the process whereby the recently pregnant uterus gradually returns to its natural nongravid state. During the involutional period, which we prefer to regard as lasting three months, the uterus shrinks from a weight of 2 pounds to a definitive weight of 1 to 2 ounces. The process of involution involves autolytic changes which lead to atrophy of individual muscle cells; the alleged mechanism of fatty degeneration which used to be held in no longer tenable.

Subinvolution is that condition in which there is a delay or arrest in the process of return to the normal non-pregnant state. However, this is not a primary disease *sui generis*. Instead, it is typically secondary to some other pathologic state. Conditions which may impede involution include: (1) retention of placental tissue; (2) imperfect exfoliation of decidua; (3) faulty involution of the placental site; (4) the presence of myomatous or adenomyomatous nodules in the uterine wall and (5) pelvic vascular engorgement which may result from uterine or adnexal inflammation or from uterine retrodisplacement.

The symptoms of subinvolution include persistent lochia rubra or frank bleeding, a sensation of pelvic heaviness and usually low backache. The diagnosis is established by bimanual examination. The uterus is found to be soft and boggy and larger than one would expect for the length of time which has elapsed since delivery and often there is an associated retrodisplacement.

The process of involution is never really complete as it is always possible to distinguish a parous from a nulliparous uterus by the demonstration of more or less elastic tissue around the blood vessels. If the perivascular elastic tissue were absorbed during the puerperium, *pari passu* with the muscular tissue, involution would really be complete; however, this is never the case and parity can always be established by histologic demonstration of perivascular collars of elastic tissue. Should the state of arrested involution become static, a type of chronic metritis known as chronic subinvolution obtains and is histologically characterized by the demonstration of excessive amounts of elastic tissue around the myometrial blood vessels. This entity has been very well described by William Fletcher Shaw[41] and by Otto Schwarz.[42] Clinically, it is manifested by a symmetrically enlarged uterus which is smooth in contour and by a history of prolonged and excessive menses dating from an antecedent pregnancy. The active treatment of subacute subinvolution cannot be overemphasized if chronic subinvolution is to be avoided. However, the pathogenesis of chronic subinvolution is indeed obscure in not a few instances and the likelihood of an endocrinopathic origin seems quite probable.

Dysfunctional Uterine Bleeding. This term implies that an aberration of physiologic mechanism rather than the existence of an organic disease is the pathogenic factor. The puerperium is all too often the setting in which dysfunctional bleeding begins. This is not surprising when one realizes the implications of the dynamics involved in the readjustment of the endocrine system following parturition or abortion.

Dysfunctional uterine bleeding can arise from any type of endometrium and usually it is anovulatory. Most functional excesses derive from an interval or estrogenic endometrium. The latter may be poorly proliferated (atrophic), normally proliferated or excessively proliferated (hyperplastic) The frequency with which cystic glandular hyperplasia of the endometrium is encountered in instances of dysfunctional bleeding arising in the late puerperium probably reflects a hangover of the anterior pituitary hyperfunction characteristic of gestation. Endometrial hyperplasia, like acromegaly, is most frequently encountered during periods of pituitary overactivity,

that is, during puberty, the climacteric and in the puerperium. Just as mild acromegaloid changes characteristic of pregnancy usually regress postpartum, although an occasional instance passes over into genuine acromegaly, similarly, postpartum hyperplasia may persist in a chronic recalcitrant state. According to Markee and Berg[43] antecedent drops of approximately 50 per cent in estrogen levels usually are associated with physiologic uterine bleeding. Moreover, there is usually a time lag of two to five days in the estrogen-bleeding relationship. Critical drops in blood estrogen content produce transient vasospasm of the spiral arterioles, with resulting ischemic necrosis of the endometrium and eventual bleeding.

Even though most cases of dysfunctional uterine bleeding observed in the late puerperium are anovulatory in origin and associated with an estrogenic endometrium, there is a distinct and not inconsiderable group associated with ovulation. In a classical paper published in 1935 Traut and Kuder[44] described "irregular shedding and irregular ripening of the endometrium" which is characterized clinically by cyclic menorrhagia, with bleeding for ten to fourteen days each month. With normal menstruation practically all the endometrium in the secretory phase is sloughed off in forty-eight hours; by seventy-two hours none is left. With irregular shedding of the endometrium, however, curettage done on the fifth or sixth day of the bleeding episode discloses secretory-phase endometrium. The glands are usually stellate in outline and the blood vessels are old (endarteritis obliterans). To establish the diagnosis curettage should be done on the fifth or sixth day of the bleeding episode. Holmstrom and McLennan[45] recently made a detailed report of three cases of irregular shedding of the endometrium: All three patients had had normal menses prior to pregnancy following which regularly recurring menorrhagia developed. Curettage was without avail in any case. The authors recommend, when necessary, hysterectomy in younger subjects and radium in older patients.

Diethylstilbestrol. More and more instances of serious secondary bleeding have been encountered in women who have received diethylstilbestrol either to inhibit lactation or to "dry up" the breasts after the establishment of lactation. Typically there is a lag of several days to a week or more between the last dose of the hormone and the onset of uterine bleeding. The bleeding may be profuse and exsanguinating. It is due to hyperestrinism and curettings typically show a pronounced estrogenic endometrium without other pathologic findings. Bimanual examination discloses no evidence of subinvolution or other abnormal findings in the uncomplicated case of hyperestinism.

The writer has often observed considerable delay in the reestablishment of ovulation and normal cyclic bleeding after stilbestrol administration in the puerperium. Whether or not an episode of serious late bleeding occurs, as previously described, often there will be a prolonged period of amenorrhea lasting for several months and not infrequently followed by a phase of metrorrhagia. It seems not unlikely that, in certain cases at least, the exhibition of stilbestrol in the puerperium can so depress the anterior lobe of the hypophysis that gonadotropin secretion is held in abeyance for an indeterminate period. From my personal experience with the use of stilbestrol in the puerperium for its alleged inhibitory effect upon lactation, I have concluded that its frequent disadvantages far outweigh its possible benefit. The all too frequent disadvantages are as follows: (1) Lactation in many cases is simply delayed and not really prevented hence, it often appears shortly after the patient's return home where the facilities for its symptomatic relief are not nearly so accessible as in the hospital; (2) the risk of secondary hemorrhage is frequent and often serious enough to require curettage and (3) prolonged amenorrhea, punctuated with episodes of profuse or extended

metrorrhagia and undue delay in the reestablishment of ovulation and normal cyclic bleeding, often occurs.

Uterine Hypotonia. Just as profuse hemorrhage may occur immediately postpartum from uterine atony, so may various degrees of uterine hypotonia in the puerperium be associated with prolonged or profuse excesses of uterine bleeding. The control of bleeding in the postpartum uterus depends upon several factors: (1) the "living ligature" action of uterine musculature in retraction and contraction; (2) venous thrombosis and (3) obliterative endarteritis. The fate of the blood vessels in the placental site has been taken up earlier in this paper.

A variety of morbid conditions may bring about diminished tone in the puerperal uterus with resulting prolonged or profuse bleeding. Patients who suffer immediate postpartum hemorrhage from uterine atony due to overdistention of the viscus from hydramnios or plural pregnancy or from uterine fatigue due to prolonged labor may require several days before regaining normal uterine tone, during which time persistent or recurrent bleeding may occur. More frequent, however, than the aforementioned instances of primary hypotonia due to muscular paralysis or fatigue are the occurrences of puerperal atony secondary to some mechanical factor which causes myometrial insufficiency. Retained placenta, myomas and adenomyosis are abnormalities which can interfere with uterine contraction and retraction. In rare cases the presence of adhesion between the uterus and surrounding organs or parietal peritoneum may prevent proper muscular contraction. According to Williams[46] atony may be associated with degeneration of the muscle fibers from an abnormal invasion of fetal elements.

The soft, hypotonic postpartum uterus may be a serious threat to the patient's life and health. Persistent oozing may go on for days or there may be a sudden profuse hemorrhage. The various factors to which hypotonia may be secondary should be carefully considered before it is assumed that the atony is primary. In either event the seriousness of the bleeding and its failure to respond to oxytocics may require timely surgical intervention including exploration of the uterine cavity, curettage, tamponade and, rarely, even hysterectomy.

Neoplasms. A variety of uterine neoplasms must be considered when inquiring into the causation of secondary postpartum hemorrhage from this source. The tumors which have been encountered as responsible for late hemorrhage include myomas, adenomyosis, endometrial polypi, choriocarcinoma, endometrial sarcoma, endometrial carcinoma and hemangioma of the uterine wall.

Myomas may cause late bleeding depending upon the location of the tumor and its pathologic stage. Subserous tumors may be excluded as a possible source of late hemorrhage. Intramural myomas may produce repeated bouts of late bleeding due to their mechanical interference with myometrial contraction and retraction, as described under uterine hypotonia. The classical cause of late bleeding from myomas appertains to submucous tumors. The latter may evoke bleeding by a variety of mechanisms: They may interfere with endometrial healing and regeneration. Pedunculated submucous myomas may bleed because of necrosis of the covering epithelium. The point of bleeding in the ordinary uterus with a submucous myoma is frequently not over the myoma itself but from the thick endometrium in some nearby furrow. Localized hypertrophic endometrium bleeds when its elongated fronds have outgrown their blood supply, with subsequent necrosis. Submucous myomas which have undergone red degeneration are particularly likely to be associated with secondary bleeding because of the tendency of red myomas to cause placenta and membranes to become glued to them. (Figs. 9 and 10.) According to Faulkner[47] red myomas, by and large, are the only ones with enough peripheral reaction to cause placenta (or

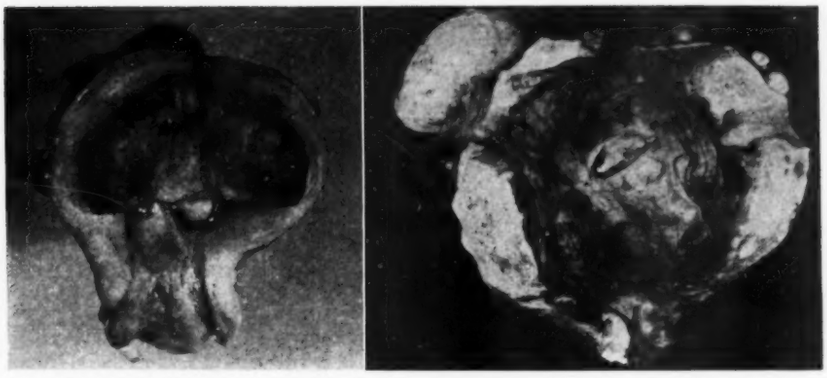

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FIG. 9. Complete uterus bisected to show large submucous myoma which has undergone red degeneration; specimen removed seventy days postpartum. Retained placenta when manually extracted after ninety-minute third stage was found adherent to submucous myoma. Curettage for delayed puerperal hemorrhage on forty-fourth postpartum day showed chronic endometritis. (See Fig. 11.)

FIG. 10. Cesarean-hysterectomy specimen removed for multiple fibromyomas; note how the membranes are adherent to a myoma showing resolving red degeneration. Red myomas are the only ones with enough peripheral reaction to cause placenta or membranes to remain adherent. (Courtesy of Robert L. Faulkner, M.D.)

bowel) to stick to them; hence the source of late bleeding in such instances may be retained placental tissue adherent to a red submucous myoma. The very rare sarcomatous degeneration of myomas may, of course, lead to delayed bleeding.

What has been said about the mechanism of delayed bleeding from the uterus with interstitial myomas applies to the dynamics in cases of internal endometriosis or *adenomyosis*. Szenes[48] has presented a classical monograph on adenomyosis as a cause of severe hemorrhage in the third stage of labor and in the puerperium.

Profuse hemorrhage may occur from *endometrial polypi* which typically have a sizeable central artery which bleeds freely when the mucosal covering is eroded. Such tumors, especially if situated in a cornu, are all too easily missed with a curette. The ordinary slightly curved uterine dressing forceps is a most useful instrument to employ after curettage for prehending an elusive polyp.

Choriocarcinoma is a malignant tumor of chorionic epithelium which may follow abortion, delivery at full-term or hydatidiform mole. It occurs about once in 30,000 deliveries. Of the seventy hydatidiform moles investigated by Brews,[49] only 8.3 per cent eventuated in choriocarcinoma. Repeated bouts of uterine hemorrhage or continual uterine bleeding is the chief symptom. The mechanism of bleeding is the erosion and invasion of uterine blood vessels by advancing sheets of tumor cells. (Fig. 11.) One must be wary in curetting uteri suspected of harboring choriocarcinoma as profuse and even fatal hemorrhage has been known to ensue.

FIG. 11. Photomicrograph of choriocarcinoma showing blood vessel almost entirely lined with tumor cells; the mechanism of bleeding is the erosion and invasion of blood vessels by neoplastic tissue. × 40. (Courtesy of Howard T. Karsner, M.D.)

A most extraordinary situation is the

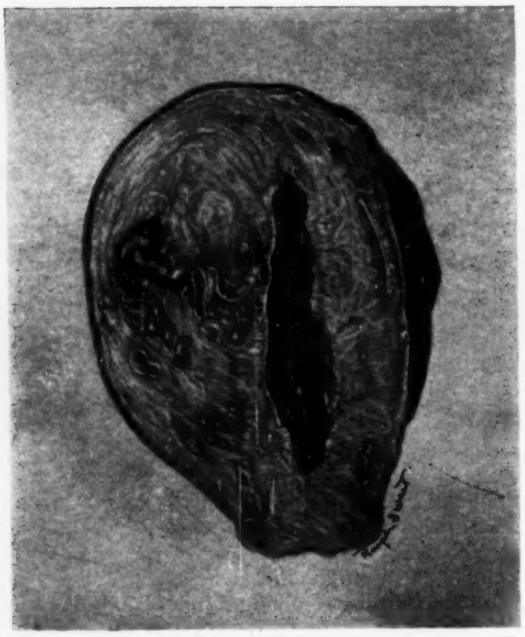

FIG. 12. Hemangioma of posterior uterine wall; patient had repeated spontaneous incomplete abortions with prolonged and excessive bleeding after each curettage. Note the great thickness of the posterior uterine wall due to the extensive hemangioma. (Courtesy of Herbert F. Traut, M.D.)

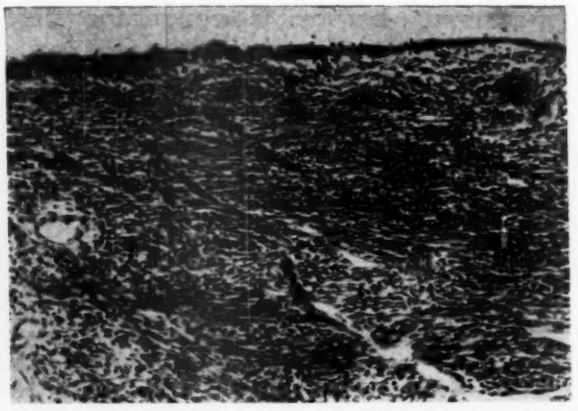

FIG. 13. Photomicrograph of curettings obtained from specimen shown in Figure 9 when patient had a severe hemorrhage on the forty-fourth postpartum day. Section shows typical chronic endometritis. The plasma cell infiltration is quite conspicuous.

coexistence of pregnancy and *endometrial sarcoma*. In 1948 Eastman[50] abstracted a case published by Stutzer[51] in the German literature. The patient experienced recurrent hemorrhages six weeks after surgical completion of an early incomplete abortion. Examination disclosed what was taken to be a myomatous uterus and hysterectomy was carried out. Scrutiny of the opened specimen showed a grossly malignant tumor, the cavity being filled with polypoid tumor masses. Microscopic study established the diagnosis of endometrial sarcoma. The patient expired six months after hysterectomy, despite postoperative roentgen therapy. Eastman refers to the two other reported cases of pregnancy associated with endometrial sarcoma. Both manifested themselves at 15 days and five weeks postpartum, respectively, by fever and uterine enlargement.

Carcinoma is surely a rare cause of late puerperal hemorrhage inasmuch as a carcinomatous endometrium is poor soil for the nidation and development of a pregnancy. However, I recall an instance of secondary hemorrhage occurring almost two weeks after salpingectomy for tubal pregnancy; investigation revealed a fairly advanced fundal carcinoma.

An excessively rare tumor is *hemangioma of the uterine wall*. Such a case was recently encountered by Dr. Herbert F. Traut.[52] The patient experienced repeated spontaneous abortions, all of which were followed by prolonged excessive bleeding despite curettage and transfusions. At the time of exploratory hysterotomy a greatly thickened and soft posterior wall was encountered. The hysterectomy specimen showed the cause of the enlargement and of the repeated postabortal hemorrhages to be an extensive hemangioma. (Fig. 12.)

Endometritis. This is really a rather rare cause of late bleeding and it is doubtless a much overworked diagnosis. When it does occur, it is much more likely to follow abortion than term delivery and its principal symptom is continued bleeding. When it follows term delivery, it is usually associated with retained secundines, submucous myomas or antecedent intrapartum infection. (Fig. 13.) As noted previously, endometritis is one of the several causes of subinvolution. One may be quite certain that unless there is something retained or present in the uterine cavity to cause and perpetuate the endo-

metritis, with the exception of tuberculous endometritis, inflammation of the endometrium does not persist for any length of time. The finding of simple endometritis on curettage in a case of late bleeding suggests that some primary entity, such as retained placental tissue or submucous myomas, probably exists but was just not demonstrated.

When endometritis is associated with chronic bleeding, as it rarely is, the underlying fault is most likely chronic myometritis, with diffuse enlargement of the uterus. The basic mechanics in such subinvolution is most likely poor contractility of the uterine muscle.

The coexistence of pregnancy and tuberculous endometritis is exceedingly rare. However, I observed this some years ago in a sixteen year old colored primigravida who was admitted to University Hospitals of Cleveland in a terminal state, with fever to 40°C., uterine bleeding and generalized sepsis, nine days after a spontaneous complete abortion at six months. The patient expired of miliary tuberculosis on her seventh hospital day, her temperature having remained between 39 and 40°C. during her hospital stay. Examination of the uterine material revealed tuberculous endomyometritis. (Fig. 14.) In retrospect it appears that the patient developed fulminating disseminated tuberculosis after conception, aborted completely and then expired from the generalized tuberculous process. Tuberculous endomyometritis was the only anatomic cause of the postabortal bleeding which could be established. Permission for necropsy was not granted.

Retrodisplacements. In speaking of secondary postpartum hemorrhage Kerr[53] states, "In a few cases I have seen a very profuse hemorrhage occur in the third or fourth week of the puerperium, when the patients were going about, and in which apparently the only cause was a backward displacement of the uterus." While it is true that retrodisplacement of the puerperal uterus may exist without associated or antecedent pathologic conditions, it is often found to be associated with subinvolution, retention of placental tissue, faulty involution of the placental site or endometritis. The mechanism of bleeding in uncomplicated cases is passive congestion or pelvic vascular engorgement; the arterial blood is delivered with the normal *vis a tergo* whereas there is impaired venous drainage. Such uncomplicated cases usually respond satisfactorily to the timely use of a properly fitting vaginal pessary, a course of oxytocics, hygroscopic (boroglycerine) vaginal suppositories and hot vaginal douches provided, of course, the cervix is closed. The use and abuse of pessaries has been taken up elsewhere.[54]

FIG. 14. Photomicrograph showing tuberculous endometritis; specimen removed because of secondary postabortal bleeding nine days after spontaneous late abortion. Note tuberculous granulation tissue and tubercle formation. Tubercle bacilli verified by acid-fast stains. × 40. (Specimen of courtesy Howard T. Karsner, M.D.)

The Postcesarean Uterus. In a consideration of the subject of secondary puerperal hemorrhage special consideration should be given the postcesarean uterus. The reason for which the cesarean was performed may predispose to late bleeding; for instance, blood-infiltrated myometrium from a case of ablatio placentae, which perhaps should have had cesarean-hysterectomy rather than simple hysterotomy, may have considerable difficulty contracting and retracting postpartum, with protracted delayed bleeding, as a result of atony. Late bleeding of a serious degree may eminate from a sinus in the lower

segment where a placenta previa had been situated. Many obstetricians have found the almost routine packing of placenta previa patients delivered by abdominal section to be a reliable prophylaxis against both immediate and delayed bleeding from lower segment and cervical sinuses. Blood loss and uterine atony create a vicious circle: The greater the blood loss, the greater the tendency to atony; contrariwise, the more profound the atony, the greater the blood loss. Hence failure to make adequate blood replacement in patients who have bled either before or after cesarean section increases the liability to postpartum hemorrhage, delayed as well as immediate.

Of particular interest is the phenomenon of delayed hemorrhage from the cesarean incision itself. Although failure to achieve adequate hemostasis in closing the hysterotomy incision usually demands attention within the first six to twelve hours postpartum, it occasionally happens that secondary bleeding will not become manifest until several days after surgery. This may be due to the breaking off of a thrombus which had temporarily maintained hemostasis or it may be due to the premature digestion of the suture material used in closing the incision. Premature solution of suture material notoriously occurs in the event of infection along the suture line. The avoidance of infection and the meticulous attention to hemostasis cannot be overemphasized.

In a report on cesarean section at the Boston City Hospital the authors[55] encountered twenty-three maternal deaths in their series of 961 cesarean sections or a percentage of 2.4. Of the twenty-three deaths one was ascribed to secondary hemorrhage on the twenty-third postpartum day. This patient had had several previous bouts of uterine bleeding before the fatal episode and, as stated in retrospect by the authors, should have had a hysterectomy early in the puerperium. Regarding the prevention of secondary hemorrhage after cesarean section the authors state, "One must be sure there is no chance for secondary hemorrhage either from uterine atony or from improper hemostasis before the abdomen is closed." Autopsy in the fatal case just reported[55] showed three large open sinuses at the placental site; the cause of death was evidently faulty involution of the placental site.

Puerperal Inversion of the Uterus. This may be a cause of serious secondary postpartum hemorrhage. Of twenty-four cases of puerperal inversion recently reported,[56] three were characterized by secondary hemorrhage. The first patient continued to bleed moderately postpartum and had difficulty in voiding. While straining in the bathroom on the seventh day the complete uterus came out of the introitus. The second patient experienced a sudden hemorrhage on the fourteenth day following a spontaneous delivery. Examination upon return to the hospital, with admitting diagnosis of "placental polyp," revealed an inverted uterus which was quite easily replaced by vaginal manipulation under anesthesia. The third patient experienced immediate postpartum hemorrhage for which transfusions were given. Despite recovery from the immediate hemorrhage the patient continued to bleed excessively and when seen by a consultant six weeks postpartum, showed a chronic inversion which, also, was replaced by taxis *per vaginam.*

Just as concealed inversion is a not uncommon cause of immediate postpartum hemorrhage, so inversion which escapes recognition at the time of delivery may later manifest itself by secondary hemorrhage. Although the fundamental cause of inversion of the uterus is not known, surely a thoughtful management of the third stage of labor is important in its prophylaxis. Forceful downward pressure on a soft fundus in an attempt to separate the placenta invites inversion.

Ruptured Uterus. Although the incidence of rupture of the uterus as encountered at University Hospitals of Cleveland[57]

was found to be only one case in every 2,756 deliveries, I believe that the occurrence of this accident is considerably more common although perhaps not recognized. Just as a certain percentage of cases of unexplained immediate postpartum hemorrhage are doubtless due to unrecognized uterine rupture, so it appears most tenable that a small percentage of cases of persistent or recurring late bleeding are associated with occult uterine rupture.

I recall two interesting cases in which the patients were delivered by internal podalic version and breech extraction. There was no immediate evidence of undue postpartum hemorrhage. However, both patients had persistent bleeding during the early puerperium with morbid courses and, after about two weeks, the development of a broad ligament abscess. Extraperitoneal drainage of the broad ligament accumulation of exudate in each case revealed an infected hematoma. In reviewing the sequence of events it appears quite patent that each patient sustained an incomplete concealed rupture of the lower segment, with extravasation of blood into the adjacent broad ligament. The advisability of immediate exploration of the uterine cavity after difficult version or breech extraction and in cases in which the operator encountered difficulty in the classical application of the anterior blade of the Kielland forceps, may have much to commend it.

Aneurysm of the Uterine Artery. Although exceedingly rare, the rupture of a traumatic aneurysm of the uterine (or coronary) artery (Fig. 15) may cause sudden exsanguinating hemorrhage. The latter may occur spontaneously or may follow the slightest straining in the puerperium. Practically any injury to the uterine artery may result in aneurysm formation. Such traumas include (1) inept forceps operations; (2) extensive cervical lacerations or incisions; (3) version and extraction; (4) breech extraction and (5) lower segment cesarean section. A cervical tear or incision which extends into the lower uterine segment (incomplete uterine rupture) may involve the uterine artery, with subsequent aneurysm formation and eventual rupture.

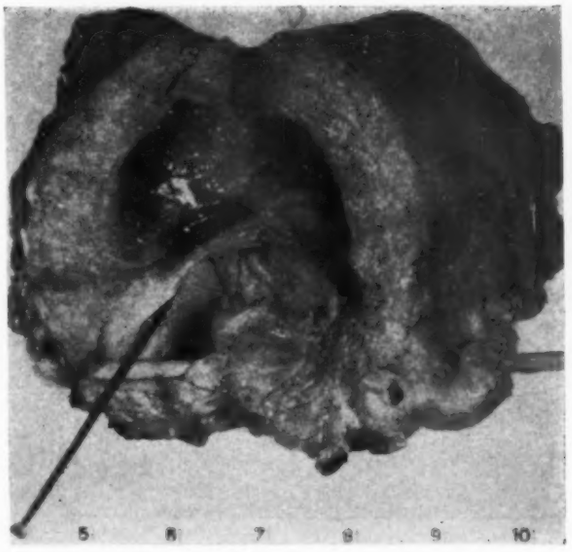

FIG. 15. Aneurysm of uterine artery; orange stick demonstrates communication between uterine artery and aneurysm sac. Late hemorrhage from uterine artery aneurysm is exceedingly rare nowadays but used to be fairly common in the era of accouchement forcé and the Bossi dilator when such were widely employed in the management of placenta previa and eclampsia. (Specimen courtesy of Herbert F. Traut, M.D.)

Vogelsanger[58] reported a case which followed version and extraction for possible placenta previa. The puerperium was uneventful for the first week; severe bleeding began on the eighth day and continued for two weeks. At hysterectomy the source of bleeding was found to be a small ruptured aneurysm of one uterine artery. Vogelsanger refers to a similar case in the literature in which hemorrhage occurred on the nineteenth and twenty-first days; at laparotomy the affected uterine artery was transfixed and recovery ensued.

The Uterine Adnexa

Late postpartum hemorrhage arising from pathologic conditions in the adnexa is exceedingly rare. However, abnormal uterine bleeding rather frequently accompanies puerperal pelvic peritonitis and adnexitis. Inflammatory involvement of the ovaries brings about interference with the normal development of the graafian follicle and

corpus luteum, with resulting erratic and frequently profuse and prolonged bleeding.

The ovarian tumors which may cause bleeding late in the puerperium are in general of two types: First, there are the corpus luteum cysts which are thought to result from the gradual resorption of blood elements in corpus luteum hematomas. Occasionally these cysts are encountered in the postpartum period. They are characteristically soft and painless and tend to fluctuate in size from one examination to the next. Once in a great while they may be the source of functional bleeding which may be so persistent as to require surgical ablation. The second group of ovarian tumors to be considered includes those which are estrogen-secreting in their physiologic behavior; although rare in the childbearing period, granulosa and theca cell tumors do occur. Although the diagnosis of granulosa or theca cell tumor is usually not really made until laparotomy, the importance of detecting and following adnexal masses cannot be overemphasized.

THE CENTRAL NERVOUS SYSTEM

This article would be incomplete without some mention of the possible role of neurogenic and psychogenic factors in the etiology of bleeding late in the puerperium.

Theobald[59] believes that vegetative control over the genital functions resides in a hypothalamic center and that menstrual abnormalities in general are reactions of this center transmitted by its neurologic connection to the hypophysis. According to Von Raisz[60] head trauma can cause hyperestrinism, with resulting endometrial hyperplasia and menorrhagia. Muller[61] studied the effect of the high altitude of a resort on young women and observed menstrual abnormalities of various sorts. Many waitresses found it necessary to return to lower levels because of serious menorrhagia. Muller hypothecated that the primary effect of high altitude is upon the hypophysis but he considered the possible influence of increased ultraviolet radiation and decreased oxygen supply upon the vessels of the endometrium. From the foregoing it is at least quite suggestive that the central nervous system is responsive to various stimuli, from head trauma to high altitude, and that a common reaction to physical agents is menorrhagia.

Miller[62] maintains that the majority of menorrhagias are psychogenic and that the chemical disturbance is secondary to a psychic factor acting on the endocrine glands. Markee[63] showed experimentally that endometrium transplanted intraocularly will show secondary hemorrhage in monkeys frightened late in the menstrual period. Emotional stress, anxiety and feelings of resentment are not infrequently expressed in the symptom of uterine bleeding. With the emotional lability and frequent psychic "let-down" so often observed in the puerperium, the stage is set for the burgeoning of certain psychosomatic responses of which abnormal uterine bleeding is not uncommon.

The psychological studies of several investigators including Novak and Harnik[64] have disclosed that the usual dynamics for abnormal uterine bleeding of psychogenic origin are a deep-seated aversion to coitus and morbid fear of pregnancy. These psychologically conditioned bleeding episodes in the puerperium are probably not dissimilar in origin to the occasionally observed premature onset of bleeding experienced by brides. Surely, women in protest against imminent biologic processes can develop a galaxy of symptoms of which unnatural uterine bleeding is not the least. However, it would be wrong to assign the cause of abnormal uterine bleeding in the puerperium to psychogenic factors by simply excluding the many organic disorders which have been found capable of evoking such bleeding; for in addition to excluding organic causes it is essential that adequate psychic dynamics be established, that is, it must be a definitely positive diagnosis.

In no specialty in the practice of medicine is psychological awareness more essential than in the discipline of obstetrics and

gynecology. To those of us who pursue the healing art even in this twentieth century the criticism voiced by Socrates[65] against the physicians of two and a half millenia ago may still be germane: "For this is the great error of our day in the treatment of the human body, that physicians separate the soul from the body."

SUMMARY

An effort has been made to review the general subject of late postpartum hemorrhage and to scrutinize the great variety of pathologic entities which may be involved in this serious obstetrical complication concerning which little has been written in a comprehensive way. The importance of considering general systemic disorders and neuropsychiatric factors as well as the gamut of purely gynecic lesions and aberrations has been emphasized. Although therapeutic suggestions appear throughout the article, it is beyond the scope of this paper to take up the management of the numerous pathologic conditions which may be involved in instances of delayed hemorrhage postpartum or postabortum. Emphasis has been placed throughout on a consideration of the natural history of the various morbid conditions, with a view to prophylaxis wherever possible; early diagnosis and, accordingly, prompt and adequate intervention. No phase of obstetric practice is more important than an appreciation of the factors involved in the care of women with late postpartum hemorrhage.

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HEXYLCAINE HYDROCHLORIDE*

A PRELIMINARY REPORT OF ITS CLINICAL USE IN COMPARISON WITH PROCAINE

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WHILE the drugs now in common use for block anesthesia are quite satisfactory, the search continues for an even better agent. Clinical trial of hexylcaine hydrochloride since April, 1948, has shown it to have sufficient promise to warrant a preliminary report. We have now used the drug as a spinal anesthetic in more than 100 patients and our experiences form the basis of this paper.

Hexylcaine hydrochloride (1-cyclohexylamino-2-propylbenzoate hydrochloride) has a molecular weight of 297.85 and is freely soluble in water. A 1 per cent aqueous solution has a pH of 4.4 (unbuffered) and is stable under conditions of boiling and autoclaving. Beyer, Latven and Freyburger[1] have compared its toxicity to that of other local anesthetic agents in laboratory animals and concluded that when injected intravenously or subcutaneously, the toxicity of hexylcaine in animals is greater than that of procaine and less than that of metycaine.

Wylde, Waters and Orth[2] have used it for spinal anesthesia in forty cases. Anesthesia was slow in onset and the level was difficult to control. This may have been due to their use of the 0.5 and 1 per cent solutions rather than the crystalline drug restored with glucose. There were no instances of irreversible nerve changes after using the agent.

In our first few cases the crystalline drug was dissolved in cerebrospinal fluid and injected in the same manner but in smaller doses than we customarily use with procaine. Because the level of the resulting anesthesia was uncontrollable and often went too high, we assumed that in some spinal fluids the drug was isobaric or even hypobaric. Weighting the drug with glucose overcame this difficulty and this method has been employed in all subsequent cases.

Our technic for single dose spinal anesthesia consists of dissolving a 50 mg. ampule of the crystalline drug in 2 ml. of 10 per cent glucose. The desired dose is drawn into a syringe and diluted with spinal fluid in sufficient amount to lower the glucose concentration to 5 to 7 per cent in the injected mixture. This is then injected subdurally through the second, third or fourth lumbar interspace, depending on the desired level of anesthesia. Our dosage has ranged from 15 mg. to 50 mg., depending on the site of the operation and the condition of the patient. In Table 1 are shown the thirty-two spinal anesthetics out of the total of 100 in which 15 to 20 mg. of hexylcaine were used. Muscle paralysis in the legs was not complete in a few cases but it was not necessary to supplement the anesthesia in any of these thirty-two cases. Rectal relaxation was excellent in the fifteen cases in which anorectal surgery was done.

In order that the reader may have a basis for comparison we have included in Table 1 the thirty-three cases from the procaine series in which 60 mg. or less of the drug were used.

We have not found it necessary to give more than 50 mg. in a single dose spinal anesthetic even for operations on the gall-

* From the Division of Anesthesiology of the Philadelphia General Hospital, Philadelphia, Pa. Hexylcaine hydrochloride was supplied under the trademark Cyclaine through the courtesy of the Department of Medical Research, Sharpe and Dohme, Inc.

bladder and stomach. We ordinarily use a total volume of 4 ml. for surgery above the umbilicus and 1 to 3 ml. below this level.

To minimize the dosage, an attempt is made to localize the injected spinal anesthetic agent around the nerve roots supplying the area in which anesthesia is desired. With this in mind the anesthetic agent is injected with the patient level. The table is then tilted slightly in the direction in which we want the solution to concentrate. In abdominal surgery it is placed in 5 to 10 degrees Trendelenburg until the level of hypesthesia can be detected at the height at which anesthesia is ultimately desired. This ordinarily takes three to five minutes although we have seen anesthesia develop in less than one minute or not appear for as long as fifteen. The table is then levelled and the height of anesthesia will be at the desired level by the time the skin has been prepared and the drapes placed. For operations on the lower extremity the solution is injected subdurally with the patient lying on the affected side. He is kept on that side for three to five minutes, or until adequate anesthesia develops, and is then placed in any position necessary for the operation.

TABLE I

CASES IN WHICH LESS THAN 20 MG. OF CYCLAINE OR 60 MG. OF PROCAINE WERE USED

Operative Site	Total No. of Cases	No. Using Small Doses	Dose		Age		Physical Status*	
			Average	Range	Average	Range	Average	Range
Cyclaine								
Anorectal	15	11	16.7 mg.	15–20 mg.	41.8	22–67	1.88	1–2
Lower extremities	46	21	16.1 mg.	13–20 mg.	65.8	50–84	3.42	3–4
Total	61	32						
Procaine								
Anorectal	27	17	50 mg.	50 mg.	32.4	21–85	1.7	1–3
Lower extremities	22	13	45.3 mg.	30–50	62.4	26–79	3.1	2–4
Perineal gynecologic	3	3	50	50	29	17–45	2.3	2–3
	52	33						

* The figures under the heading Physical Status refer to those given by the American Society of Anesthesiologists in categories 1 to 4 in which 1 is the best physical status and 4 is very poor physical condition.

TABLE II

AGE GROUPS

SHOWING AGE DISTRIBUTION OF PATIENTS GIVEN SPINAL ANESTHESIA WITH PROCAINE AND WITH HEXYLCAINE

Age	Hexylcaine	Procaine
10–19	7	12
20–29	10	17
30–39	16	20
40–49	17	22
50–59	21	12
60–69	14	11
70–79	12	5
More than 80	3	1
	100	100

As a basis for comparison we have taken 100 cases of spinal anesthesia in which procaine was used as the agent. These cases were picked at random from our Chicago Keysort[3] files during the same period of time in which we were trying hexylcaine. By taking the same period for the two series we believe the variables such as surgeons, operative technics and anesthesiologists will be equalized.

In Table II we have shown the age distribution in the two groups of patients to indicate that the patients in the two series were comparable. The average age of the

patients given procaine spinal anesthesia was 42.5 years while that of those given hexylcaine was 48.5.

Additional intravenous vasopressor for spinal hypotension was required by 12 per cent of the procaine group and by 21 per cent of the hexylcaine series. Although we have no means of measuring this, we have been interested to hear several of the surgeons remark on the completeness of the abdominal muscle paralysis which spinal anesthesia with hexylcaine provides. Epinephrine, 0.5 mg., was added to the spinal anesthetic solution to prolong the anesthetic time in 56 per cent of the procaine series and 7 per cent of the hexylcaine series. In order to complete the operation, supplementary agents other than dilute pentothal were added to 6 per cent of the procaine group and to 2 per cent of the cyclaine series.

In Table III we have grouped the 100 cases in each series according to the region of operation. It will be noted that the dose of hexylcaine was in most cases about one-third that of procaine. Due to the pressure of clinical work it has not been possible to follow-up all the patients on the wards after operation to find out exactly when the spinal anesthesia wore off. It is our impression from observation in the operating rooms and on the wards that in the doses given spinal anesthesia with hexylcaine lasts sixty to ninety minutes.

TABLE III
COMPARISON OF PROCAINE AND HEXYLCAINE

Regions	Procaine				Hexylcaine			
	No. of Cases	Average Dose mg.	Average Duration Minutes*	Average Physical Status‡	No. of Cases	Average Dose mg.	Average Duration Minutes*	Average Physical Status‡
Upper abdominal								
1. Gallbladder	1	150	115	3	1	50	40	3
2. Stomach duodenum	1	120	65	3	1	50	55	4
3. Other	1	120	0†	4	1	40	45	2
Lower abdominal								
1. Appendix	12	135.8	66.8	1.6	10	50	49.5	1.7
2. Bowel	1	150	60	2.0				
3. Obstetric operations	1	120	80	3				
4. Gynecologic operations	5	132	77	2	6	50	85	1.8
5. Others	2	135	48	3	3	50	75	2.7
Abdominal wall								
1. Inguinal	19	140.5	64	1.5	12	48.3	65.8	1.66
Perineal								
1. Anorectal	27	65.5	31.1	1.6	15	19.6	33.8	1.7
2. Perineal genitourinary	5	131.0	59	2.0	2	31	60	2.5
3. Perineal gynecologic	3	67.5	34.5	2.5	3	37.6	83.3	2
Limbs								
1. Lower soft	4	98.7	49	1.2	9	21.7	48.0	2.7
2. Lower bones	18	59.3	38.5	3.1	37	24.7	41.4	2.0

* Duration is from injection of anesthesia to completion of operation
† Spinal failure
‡ See footnote Table 1

We have also used hexylcaine in fifteen fractional spinal anesthetics with the catheter technic described by Touhy[4] and the modification for segmental spinal anesthesia described by Saklad et al.[5] Our present method is to dissolve 150 mg. of hexylcaine crystals in 6 ml. of 10 per cent glucose and dilute the mixture to a volume of 10 ml. with spinal fluid. This makes a concentration of 1.5 per cent or 15 mg. per

ml. We use an initial dose of 15 to 30 mg. and usually give 15 mg. every twenty to thirty minutes after the first half hour. The smallest dose by this technic was 30 mg. for a ninety-minute supravaginal hysterectomy in a forty-six year old patient; the largest dose was 135 mg. in two hours thirty-five minutes in a fifty-two year old patient for exploration of the common bile duct. By comparison, a 100 minute supravaginal hysterectomy in a sixty-two year old patient required 250 mg. of 5 per cent procaine and a colectomy in a sixty-two year old man required 500 mg. of 5 per cent procaine in two hours fifty minutes.

In addition to using hexylcaine for spinal anesthesia we have employed it in over 200 regional and local nerve blocks in concentrations varying from ¼ to 1 per cent. Our impression to date is that it is more effective in weaker concentrations than procaine is. This is in keeping with our results when the drug is used in spinal anesthesia. In a recent brachial block in which 30 ml. of 1 per cent hexylcaine without epinephrine were used, the anesthesia lasted five hours. Following intracutaneous or deep injection some patients complain of mild burning for a short period immediately after injection.

In our experience with this drug to date we have not seen a local or general toxic reaction. There have been no instances of irreversible changes in nerve function.

SUMMARY AND CONCLUSIONS

The use of hexylcaine hydrochloride in 100 single dose spinal anesthetics and fifteen fractional spinal anesthetics has been described. The results have been compared with those of two similar groups in which procaine hydrochloride was used. The use of hexylcaine in local and regional block anesthesia has been mentioned.

Our experience to date indicates that this new drug provides more complete and longer lasting nerve block in smaller doses relative to its animal toxicity than does procaine. For these reasons we believe further clinical trial is warranted. We are continuing our studies and hope that others will try it so that the large amount of data required for the accurate appraisal of a new drug will be amassed.

ADDENDUM

Addendum. Since this manuscript was submitted for publication, hexylcaine hydrochloride has been successfully used in 400 additional spinal anesthetics. The conclusions reached in this preliminary report remain unchanged by this more extensive experience.

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DUPLICATION OF THE SMALL INTESTINE*

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ALTHOUGH considered a rare anomaly duplication of the bowel probably occurs more frequently than has been suspected. Donovan has operated upon six patients in eighteen months and regards it as one of the important causes of massive intestinal hemorrhage in infants. Since almost all duplications produce symptoms of an acute nature, it is essential that the surgeon be familiar with the condition and be prepared to treat it.

Etiology. The etiology of intestinal duplication has been the subject of much controversy and many theories have been put forward to explain the anomaly.

Any explanation to be acceptable must account for the fact that duplications occur at all levels of the intestinal tract and that they are always on the mesenteric side of the true bowel situated between the leaves of the mesentery. This rules out the idea that these anomalies are of the same nature as Meckel's diverticulum and are due to persistence of the proximal part of the vitelline duct. In addition cases have been reported by Edwards and by Grove and Porch of duplication occurring when a Meckel's diverticulum was also present on the antimesenteric border of the bowel.

Sequestration of the intestinal epithelium has been suggested by Hughes-Jones as a possible etiologic factor. Lewis and Thyng have demonstrated diverticula occurring along the mesenteric border of the embryonic intestine of various mammals and man. Normally these disappear as development continues but they may persist and be pinched off from the bowel. This explanation accounts for spherical duplications and mesenteric cysts but it is difficult to see how the long, tubular variety could be produced in this manner.

The entire subject has been thoroughly reviewed by Bremer who considers that the spherical duplications are best explained by the theory of Lewis and Thyng but that the tubular forms are derived from abnormalities in the development of the solid stage of the intestine. In the six-week embryo the intestinal tract undergoes a period of rapid growth and the epithelial cells proliferate to fill the lumen forming a solid cord. This may occur only in isolated regions of the tract. Normally, as growth progresses, vacuoles appear in the solid cord; these fuse to form a lumen. This process has been well demonstrated by Johnson in wax models of the developing intestine. It is easy to see how, by abnormalities of fusion, two, three or even four lumina might be formed. As growth continues, the lumina are separated; and the undifferentiated cells surrounding the epithelium give rise to all the coats of the bowel. This theory explains all the variations thus far reported in clinical cases of intestinal duplication. Atresia and stenosis of the intestine can be accounted for in a similar manner by failure of vacuolization and the persistence of a solid cord for varying lengths of the tube. Duplication is occasionally associated with atresia of the normal bowel, as in Case 1 of the present series, and it is possible that a similar stimulus may initiate both abnormalities. (Fig. 1.)

Pathology. Duplication of the intestine may occur anywhere in the tract but it is most common in the ileum. The duplicate bowel lies along the mesenteric border of

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the intestine between the leaves of the mesentery.

The duplicate lumen may communicate with the intestinal tract at one or both ends or not at all. Communication usually occurs at the distal end and proximally the duplication ends as a blind, rounded tube. It may vary in length from 1 to 120 cm. and it may occasionally exist as several isolated tubular structures lying free within the leaves of the mesentery. (Fig. 2.)

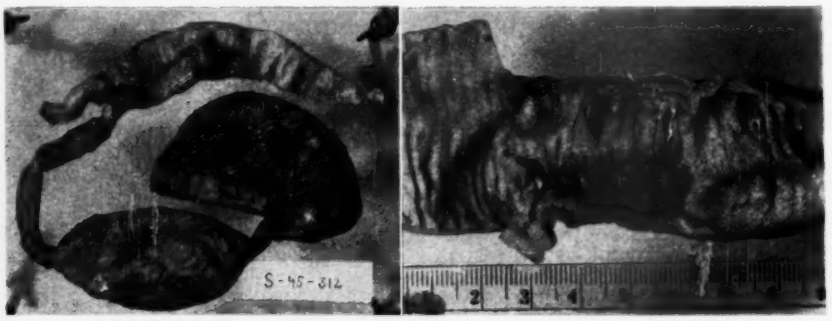
A B C

FIG. 1. Diagram of development of duplication of intestine; A, solid stage; B, vacuolization and C, complete duplication.

The structure of the duplication is that of normal bowel. The mucous membrane may be gastric or intestinal and the level of the anomaly has no influence on the type of epithelium present. Ectopic gastric mucosa is frequently found.

In some cases the superficial epithelium resembles that of the normal intestine but gastric glands with acid-secreting cells are found in the deep layers. In others the duplication is lined with typical gastric mucosa and sections resemble the stomach. This type may secrete true gastric juice containing the enzymes pepsin and rennin as well as free hydrochloric acid.

The reason heterotopic mucosa develops in these anomalous structures is as yet unknown. Taylor has contributed a complete pathologic description of this interesting condition but, as Edwards points out, he underestimates its clinical significance. In this series and in all the others reported ectopic gastric mucosa has usually been associated with peptic ulceration of the intestine at the point of communication. This may lead to the complications of massive hemorrhage or perforation with peritonitis. (Figs. 3 and 4.)



2 3

FIG. 2. Specimen of duplicate ileum resected; note the large stomach-like pouch. Case 11.

FIG. 3. Duplicate bowel showing peptic ulceration of mucosa. Case 11.

CLINICAL FEATURES

Symptoms due to duplication of the intestine commonly occur in infancy or early childhood. In the series reported by Ladd and Gross the age incidence was from two weeks to nine years. Occasionally symptoms begin at birth. The clinical picture can best be considered under the following headings:

Intestinal Obstruction. This may be caused by several factors, namely, by pressure from the increasing size of the dupli-

cation, intussusception and volvulus; the duplication may be associated with intestinal atresia. All of these have been reported in the literature. A palpable mass is usually present and x-ray of the abdomen may show compression of the bowel by an abnormal gas-filled shadow.

Symptoms Due to Peptic Ulceration. Intermittent abdominal pain has been reported by Edwards in a case which ultimately perforated necessitating an emergency laparotomy. Diarrhea due to acid irritation of the ileal mucosa occurs quite frequently. Massive hemorrhage is not uncommon. Duplication should always be considered in the differential diagnosis of intestinal bleeding in infants. The surgical procedure involved in dealing with these cases is so serious that exploration should not be attempted until the child has been transfused and brought back to as good a condition as possible. Perforation with local abscess or general peritonitis is a rare complication but has been observed by Edwards and by Black and Benjamin.

TREATMENT

The treatment of choice is excision. This always involves resection of the adjacent bowel because the duplication lies between the leaves of the mesentery and cannot be removed without compromising the circulation of the normal intestine. Side-to-side anastomosis is technically easier and safer than end-to-end.

Short-circuiting operations and the creation of windows between duplication and bowel lumen have been tried but usually fail to relieve the symptoms. If gastric mucosa is present, ulceration occurs at the site of anastomosis and symptoms recur. The only effective treatment is complete removal of the anomaly. The difficulties and dangers of bowel resection in infants are well recognized but with careful preoperative management and massive transfusion the procedure does not carry an excessive mortality. Donovan reports six cases in which all patients survived resection. This article reports on an additional four patients, three of whom survived operation.

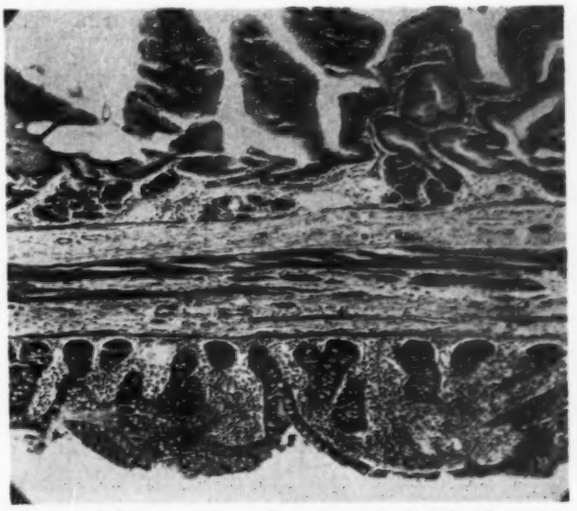


FIG. 4. Microscopic section through duplication; the two lumina have a common muscular wall with ileal mucosa above and gastric mucosa below on the side of the duplication. Case 11.

CASE REPORTS

CASE 1. F. W. age one day, was admitted to the Children's Memorial Hospital June 25, 1945. This baby was born three hours prior to admission. Abdominal distention was noted at birth and a flat plate of the abdomen showed evidence of small bowel obstruction. The anus was patent. Clinical examination was otherwise negative. Laparotomy was performed eighteen hours post partum and a duplication of the terminal ileum was found which had formed a volvulus around its blind, pouch-like ending. The gangrenous bowel was excised and a side-to-side anastomosis performed. An ileostomy was done to relieve distention. The child died eight hours postoperatively.

The pathologic report (supplied by Dr. F. W. Wigglesworth) revealed that the specimen consisted of a dark, greenish blue length of bowel which was small in diameter at one end and dilated at the other. There was a hemorrhagic, fibrous mesentery attached throughout its entire length and at one point contained what appeared to be another narrow piece of bowel which was curled up on itself. It did not communicate with the true lumen. Sections of the true bowel and the duplication showed normal small bowel structure with intestinal mucosa. The diagnosis was duplication of ileum with volvulus.

CASE II. C. C., age three months, was admitted to the Children's Memorial Hospital November 8, 1945, with the complaint of passage of blood by rectum two weeks prior to admission.

Clinical examination was negative and an exploratory laparotomy was undertaken with a preoperative diagnosis of bleeding Meckel's diverticulum. A duplication of the terminal ileum was found which was resected and the bowel continuity reestablished by a side-to-side anastomosis.

The postoperative course was uneventful. The patient recovered and was discharged from the hospital free from symptoms.

The pathologic report (supplied by Dr. F. W. Wigglesworth) revealed that the specimen consisted of a length of ileum measuring 20 cm. At a point 7 cm. from the distal end it was seen to bifurcate into two portions at its mesenteric border. These were closely applied and ran parallel to one another for a distance of 8 cm. At that point they separated. The duplication continued 11 cm. further, gradually narrowing at its proximal end. Here it widened to form a large fusiform pouch which was symmetrical and measured 10 cm. in length by 5 cm. at its widest point. The distal end of the pouch gave rise to a short narrow portion 3 cm. in length. This opened into another large, purse-like pouch which formed the most proximal part of the diverticulum and ended blindly.

Two ulcers were present in the ileum-like portion situated on the wall which was in apposition to the bowel. The distal pouch contained smooth ileal mucosa with two raised islands of white gastric mucosa. The proximal pouch was lined with gastric mucosa and showed three peptic ulcers.

Fluid aspirated from this proximal pouch showed a free acidity of 30, total acid of 60 and the presence of pepsin and rennin. The diagnosis was duplication of terminal ileum with accessory stomach.

CASE III. E. P. age two and one-half months, was admitted to the Royal Victoria Hospital April 18, 1947, following a massive intestinal haemorrhage one hour prior to admission.

The past history was negative except for sporadic diarrhea for two weeks before onset of the hemorrhage. On admission the child was pale, cold, and almost exsanguinated; the diaper contained a great deal of dark red, clotted blood. Physical examination was otherwise negative. Blood transfusion was started at once. The child's condition improved remarkably in the next few hours and he was taken to the operating room where under ether anesthesia exploratory laparotomy was performed by Dr. C. A. McIntosh. A reduplication of the terminal ileum was found with the duplicate bowel extending from a point 12 cm. proximal to the ileocecal valve to the jejunum, a total length of 90 cm. The duplicate bowel communicated with the normal lumen at the distal end and at this point a hard, indurated, ulcerated area could be felt.

Because of the child's precarious condition and the great length of the duplication, resection of the entire anomaly was considered too hazardous a procedure. A segment of normal and reduplicated bowel 35 cm. in length was excised. This contained the indurated area and it was hoped that enough gastric mucosa was removed to prevent further ulceration. The intestinal continuity was reestablished by side-to-side anastomosis and enteroanastomosis was performed between the duplication and normal bowel to prevent formation of a blind loop.

The patient's postoperative course was satisfactory for ten days and he was taking fluids well. On April 29th he had a moderate melena and on April 30th there was a fairly massive hemorrhage from the bowel. It was considered that further ulceration had occurred and a more extensive resection was necessary.

On April 30th under intratracheal cyclopropane anesthesia an operation was performed by Dr. C. B. Ripstein. The remaining duplication and the adjacent ileum was resected (55 cm.) and a side-to-side ileocecostomy performed. The immediate postoperative course was satisfactory. Penicillin and streptomycin were given and continuous gastric suction used for forty-eight hours. The patient was then put on feedings of breast milk and took these without vomiting. He began to pass frequent, watery stools on the third postoperative day and continued to pass from four to six stools daily. These contained digested food material and no blood. Hydration was maintained with difficulty by frequent clyses of normal saline and plasma. Despite adequate oral intake supplemented by parenteral feedings and vitamins the child lost weight steadily.

On May 12th the diarrhea became more profuse and the temperature was elevated to 100°F. Culture of the stools revealed a profuse

growth of salmonella. Streptomycin was given and the electrolyte and water balance maintained by intravenous infusions and the child improved. However, his nutrition could not be maintained. Over the period of the next month his condition gradually deteriorated and his weight dropped to 8 pounds from an initial weight of 11 pounds 3 ounces. The child expired June 28, 1947, from a terminal staphlycoccal bacteremia and septicemia.

Postmortem examination showed extreme inanition. The residual small bowel measured 60 cm. in length.

The pathologic report (supplied by Dr. T. R. Waugh) revealed that the surgical specimen consisted of 90 cm. of small bowel. Seven cm. above the distal end of the specimen the bowel bifurcated and became a double structure; the smaller loop coursed within the mesentery of the larger, the junction between the two being on the mesenteric border of the larger loop.

Immediately beyond the point where the two loops joined the wall of the ileum was thickened and there was an irregular ulcer 1 by ½ cm. with indurated, heaped up margins.

Sections through the two lumina at the point farthest from their juncture showed that the ileum consisted of a thin walled structure with normal architecture and lined by glands showing the usual pattern. The duplication showed a mucosa consisting of tall, superficial villous folds lined by columnar epithelium and resembling those of normal ileum. The deeper part of the mucosa contained tubular glands similar to those found in gastric mucosa and lined with numerous acid cells.

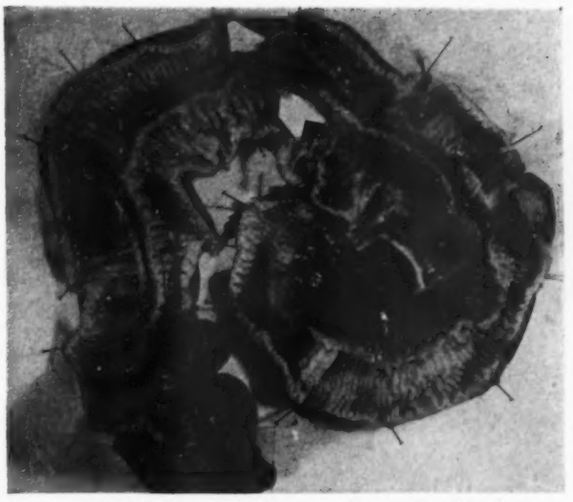
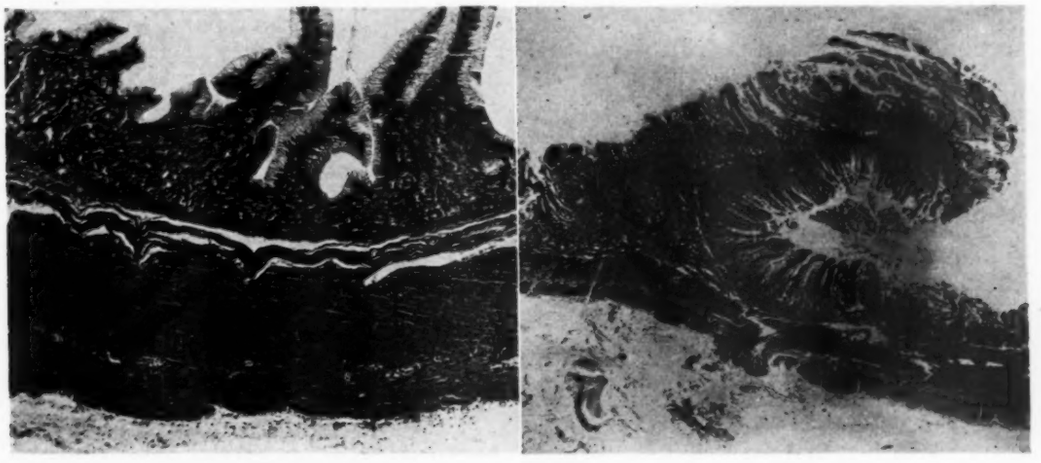


FIG. 5. Specimen resected; complete reduplication on mesenteric side of ileum. Case III.

Sections through the point of juncture of the two loops showed a spur covered on one side by the typical gastric mucosa with glands containing many acid cells. This was abruptly replaced at the apex of the spur by normal ileal mucosa which showed peptic ulceration and diffuse infiltration with round cells, polymorphonuclears and eosinophiles. The diagnosis was duplication of the ileum lined by heterotopic gastric mucosa and peptic ulcer distal to junction. (Figs. 5, 6 and 7.)

CASE IV. D. D., age six days, was admitted to the Children's Memorial Hospital, June 9, 1947. This child was born June 2, 1947, and vomited immediately after birth. Blood and



6 7

FIG. 6. Section through duplication; the mucosa shows superficial intestinal epithelium and villi with deep, gastric-type glands. Case III.

FIG. 7. Section through spur at juncture of true and duplicate lumina; transition between ileal and gastric mucous membranes. Case III.

mucus were passed in the meconium in the following three days and on examination a mass was felt in the abdomen. X-ray examination showed evidence of obstruction in the upper small bowel and laparotomy was performed.

At operation a volvulus of the jejunum associated with a duplication was found 17 inches from the duodenojejunal junction. In addition a fibrous atresia of the jejunum was present. The volvulus was resected and intestinal continuity restored by side-to-side anastomosis. The patient's postoperative course was somewhat stormy but the child made an eventual complete recovery.

The Pathologic report (supplied by Dr. F. W. Wigglesworth) revealed that the specimen consisted of 30 cm. of jejunum. Three cm. from the proximal end there was a narrowing of the lumen with a diameter of 0.2 cm. Between the layers of the mesentery there was an oval, cyst-like duplication which did not communicate with the intestine. Microscopic sections showed that the duplication contained all coats of normal bowel but the epithelium was flattened and its structure indefinite. The diagnosis was duplication of jejunum with volvulus.

COMMENT

These cases represent fairly typical examples of the clinical pictures produced by duplication of the small bowel. Bleeding was present in three cases and intestinal obstruction in two.

Case III presents several interesting problems. The child survived operation immediately but the resection was too extensive (90 cm. of small bowel) to permit proper nutrition and the child ultimately died of inanition complicated by secondary bacterial infection. Digestion was normal but absorption was inadequate and the technical difficulties of long-continued parenteral feedings in a three month old baby made it impossible to maintain life longer than two months. Donovan reports a case in which the patient survived resection of 45 cm. of ileum but no reports could be found of any more extensive resections in infants.

Case II has been carefully studied by Dr. F. W. Wigglesworth, pathologist to the Children's Memorial Hospital, Montreal. His observations on the acid and enzyme content of the juice aspirated from the duplication present a unique contribution to our knowledge of the pathologic physiology of this anomaly.

SUMMARY

1. Four cases of duplication of the small intestine are presented; successful resection was carried out in two. One patient survived operation immediately but died of inanition due to lack of intesting absorption and the fourth patient died eight hours postoperatively.

2. The etiology, pathology and clinical features of the anomaly are reviewed and the fact is reemphasized that it must be considered in the differential diagnosis of intestinal hemorrhage and obstruction in infants.

I would like to record my thanks and appreciation to Drs. C. A. McIntosh, A. K. Geddes and D. E. Ross for their help in the preparation of this paper.

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SPINAL ANESTHESIA UTILIZING PONTOCAINE IN HYPOBARIC SOLUTION FOR SURGICAL PROCEDURES ON THE ANORECTAL REGION*

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ANESTHESIA for surgical procedures in the anorectal region often presents a problem to which there is no entirely satisfactory solution. Various technics of general and regional anesthesia have been employed by anesthetists and surgeons in attempts to overcome the difficulties inherent in the prone position, namely, flexion of the patient, the necessity of providing both sensory anesthesia and relaxation of the anal sphincter, the maintenance of efficient circulatory and respiratory function and, on a busy surgical service, the accomplishment of these tasks with a minimum expenditure of time. It is the purpose of this communication to redirect attention to a technic, previously described by Lund and Rumball[1] but insufficiently utilized by the profession, that may offer an approach to the solution of these problems. The present series of spinal anesthesias utilizing pontocaine in hypobaric solution for surgical procedures involving the anorectal region is far too limited numerically to permit statistical evaluation. The results to date have been encouraging, however, and suggest that the method warrants further investigation.

The extent of spread of an anesthetic solution within the confines of the subarachnoid space is governed by a number of factors.[2] Of these the controllable factors that are of most importance in limiting the extent of anesthesia are the difference in specific gravity between the injected solution and the cerebrospinal fluid and the position of the spinal canal at the time of injection. These facts have been well demonstrated by Sise,[3] employing hyperbaric solutions, and both Etherington-Wilson[4] and Jones,[5] employing hypobaric solutions. Lund and Cameron[6] have utilized pontocaine in hypobaric solution for spinal anesthesia, employing what they have termed a modified Howard Jones technic. More recently Lund and Rumball[1] reported the use of spinal anesthesia utilizing pontocaine in hypobaric solution for dealing with surgical procedures on the posterior aspects of the lumbar and sacral regions. They refer to this method as the prone technic since the patient is placed prone and flexed at the hips and the subarachnoid injection is performed in this position. Lund[7] now adds small quantities of a vasoconstrictive substance (epinephrine or neosynephrine) to the pontocaine solution in order to obtain more prolonged anesthesia but this should certainly be unnecessary for the type of surgical procedure to be discussed in this communication.

The theoretical advantages accruing from the use of this prone technic of spinal anesthesia utilizing pontocaine in hypobaric solution for surgical procedures on the anorectal region are numerous. The patient is in the optimal position at the time that lumbar puncture is performed and no additional time must be spent in placing the patient in position for the surgical procedure. The rapid onset of sensory analgesia allows the surgical intervention to be begun immediately. The definite limitation of the extent of the anes-

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FIG. 1. The patient is placed prone upon the operating table and then flexed 30 to 40 degrees at the hips. A pillow, hidden by drapes in the illustration, is put under the pelvis and lower abdomen to reduce the normal lumbar lordosis. Lumbar puncture is then performed.

thesia to the lumbosacral dermatomes ensures an absence of circulatory depression in the majority of instances. Adequate anal sphincter relaxation is assured by the efficient concentration of the anesthetic solution in the area providing motor innervation to the sphincters. Motor power to the lower extremities remains intact due to the limitation of anesthesia, permitting immediate ambulation upon completion of the surgical procedure, thereby minimizing postoperative vascular and respiratory complications.

These various considerations prompted a limited investigation of Lund and Rumball's prone technic of spinal anesthesia utilizing pontocaine in hypobaric solution for surgical procedures on the anorectal region.

TECHNIC

The patient is placed prone upon the operating table and then flexed 30 to 40 degrees at the hips. (Fig. 1.) A pillow under the pelvic brim and abdomen aids materially in reducing the normal lumbar lordosis and thus facilitates the performance of lumbar puncture. The actual insertion of the needle into the subarachnoid space is performed very slowly and carefully to enable the anesthetist to feel the needle traverse the dura. When the needle is felt to "click" through the dura, the stilet is withdrawn and the patient is asked to contract the abdominal muscles; this straining often will force cerebrospinal fluid out of the needle. Sometimes even this maneuver will fail to produce fluid even though the anesthetist may be certain that the needle is properly placed within the subarachnoid space. On such occasions aspiration with an empty syringe may reveal cerebrospinal fluid and confirm the proper position of the needle. Lumbar puncture is performed with the bevel of the spinal needle turned laterally but the bevel is turned caudad prior to subarachnoid injection of the anesthetic solution. The performance of lumbar puncture in the prone, flexed position is the major difficulty encountered in carrying out this technic, and it is difficult, some experience

being required before it can be performed with facility.

One-tenth per cent pontocaine solution is prepared by withdrawing 1 cc. (10 mg.) from a 2 cc. (20 mg.) ampule of 1 per cent pontocaine and adding to it 9 cc. of triple-distilled water withdrawn from a 10 cc. ampule. It is imperative that these measurements are absolutely correct and that the two fluids are then properly mixed. The solution may also be prepared by adding triple-distilled water to pontocaine crystals. Recently 0.1 per cent pontocaine solution was made available for research purposes in 20 cc. ampules containing 20 mg. of pontocaine. The specific gravity of cerebrospinal fluid varies between 1.004 and 1.006 under conditions of normal health.[8] The specific gravity of 0.1 per cent pontocaine solution is 1.002 at room temperature (24.4°C.). The dose employed varies with the extent of the surgical procedure. (Table 1.) As little as 4 mg. of pontocaine may be sufficient for the removal of external hemorrhoidal tabs whereas 8 mg. of pontocaine may be required for an extensive excision of a large pilonidal sinus. Injection of the anesthetic solution is performed very slowly (10 to 20 seconds) at an even rate in order to prevent undue mixing with the cerebrospinal fluid and to allow the anesthetic solution to float along the posterior aspects of the subarachnoid space.

RESULTS

Spinal anesthesia utilizing pontocaine in hypobaric solution was employed successfully in seventy-five instances for surgical procedures on the anorectal region and was attempted but abandoned in five other instances. The average patient was a healthy, young, adult male veteran. Therefore, there was little opportunity in this series to evaluate the use of the technic in poor risk patients requiring this type of surgical intervention. Four patients were in the second decade of life, forty-eight in the third, sixteen in the fourth, three in the fifth and four in the sixth. The sex distribution within the series was unequal, there being seventy-four male patients and one female patient.

The types of operation performed, their relative incidences and the dosages employed are shown in Table 1. It should be noted that hemorrhoidectomies and excisions of pilonidal cysts and sinuses made up the great bulk of the surgical procedures. The time necessary to mix the hypobaric solution of pontocaine, perform lumbar puncture and administer the anesthetic solution is shown in Table II. The average time necessary to accomplish these duties was six minutes which approximates the time necessary for the administration of a spinal anesthetic with the patient in either a lateral or sitting position. The time necessary for the onset of analgesia after the administration of the anesthetic solution is shown in Table III. The average time was three to four minutes which is considerably less than that necessary for the onset of analgesia and fixation of the intrathecally-placed anesthetic solution be-

TABLE 1
CORRELATION OF DOSAGE EMPLOYED WITH THE TYPE OF OPERATIVE PROCEDURE

Operative Procedure	No. of Patients and Dosage Employed					
	4 mg.	5 mg.	6 mg.	7 mg.	8 mg.	Total
Hemorrhoidectomy	2	8	19	1	1	31
Fistula-in-ano	1	4	5	1	0	11
Pilonidal cyst/sinus	0	4	16	6	2	28
Perirectal abscess	0	0	3	0	0	3
Miscellaneous	1	0	1	0	0	2
Total	4	16	44	8	3	75

TABLE II
TIME REQUIRED FOR THE ADMINISTRATION OF ANESTHESIA

Minutes	No. of Patients
3	7
4	7
5	11
6	22
7	10
8	6
9	4
10 or more	8
Total	75

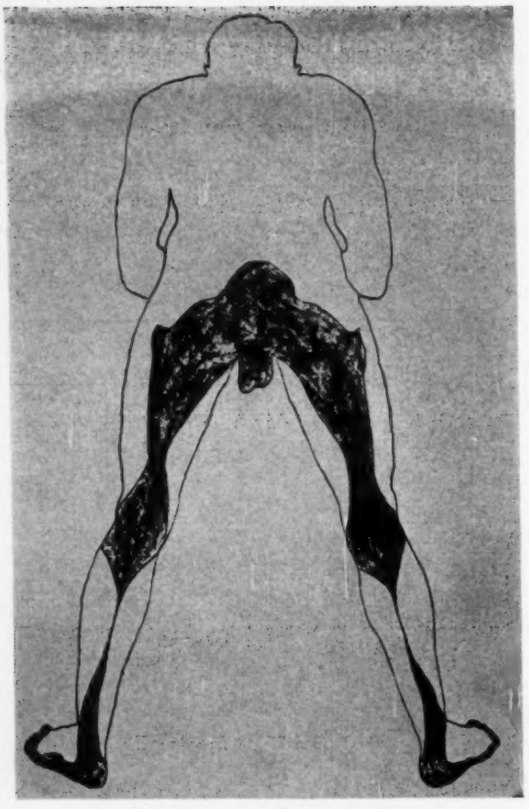

FIG. 2. Area of anesthesia.

fore the patient can be positioned and surgery begun after the ordinary spinal anesthetic. This constitutes an important advantage of the hypobaric pontocaine technic since the patient is already in the optimal position and the surgical procedure may be begun immediately following the onset of analgesia.

TABLE III
TIME OF ONSET OF ANALGESIA FOLLOWING THE ADMINISTRATION OF ANESTHESIA

Minutes	No. of Patients
2	14
3	26
4	25
5	6
6	1
7 or more	3
Total	75

The extent of the anesthesia was usually limited to the "saddle-seat" area. (Fig. 2.) In the majority of instances the upper level of anesthesia was below the fifth lumbar dermatome and some motor function was retained in the lower extremities throughout the operative period. The larger doses of pontocaine in hypobaric solution (6 to 8 cc.) raised the level of anesthesia by one or two dermatomes. However, a change in the position of the patient resulted in a marked change in the level of anesthesia

TABLE IV
DURATION OF ANESTHESIA

Minutes	No. of Patients
90	6
120	20
150	15
180	25
210	4
240 or more	5
Total	75

and warrants a word of warning. The patient should be definitely and repeatedly admonished not to raise his head or shoulders until at least twenty minutes after the administration of the anesthetic solution. On two occasions patients failed to adhere to this caution and raised their heads and shoulders off the table for a short period of time. The level of anesthesia ascended rapidly into the mid-dorsal region. On two other occasions the level of anesthesia appeared to be too low for the excision of extensive pilonidal cysts but ascended into the upper thoracic region when the patients' heads were raised for short periods of time.

The duration of anesthesia (Table IV) averaged two or more hours. Occasionally anesthesia of sufficient intensity to permit an operative procedure was still present four hours after the administration of the hypobaric solution. A definite correlation could be established in many instances between a given dose of pontocaine and the duration of anesthesia, namely, 6 mg. of pontocaine produced anesthesia lasting from two and a half to three hours whereas 4 mg. of pontocaine produced anesthesia lasting only from one and a half to two hours.

There were five instances in which it was necessary to abandon the procedure as described and utilize some other method of anesthesia. In four such instances it proved

to be impossible to perform lumbar puncture in the prone-flexed position; this undoubtedly was due to the anesthetist's inexperience with the technic. The fifth failure occurred in a twenty-six year old, male negro who was scheduled for a re-excision of condylomata acuminata of the anal region and in whom the original excision had been successfully performed under spinal anesthesia administered in the prone-flexed position. Lumbar puncture was again successfully performed in the prone-flexed position; however, the surgical service requested a 10 cc. sample of cerebrospinal fluid for analysis and it proved to be impossible to obtain this large an amount. The patient was therefore turned to the lateral decubitus position, a second lumbar puncture as performed, the necessary cerebrospinal fluid was obtained and the spinal anesthetic was then administered.

Certain complications arose during the course of operation and were attributed to the anesthetic method. There were eight instances in which the patient, during the course of hemorrhoidectomy, complained of a dull, aching, abdominal pain often limited to the suprapubic region but occasionally referred to the epigastrium. This pain occurred simultaneously with overdilatation of the rectum and promptly disappeared when such dilatation was discontinued. This was undoubtedly referred pain travelling through visceral afferent fibers of the sympathetic nervous system to the hypogastric and aortic plexuses. In only one instance was it necessary to administer supplementary anesthesia; all the other patients described the sensation as being annoying rather than truly painful. In three instances the level of anesthesia was inadequate for the extent of the operation contemplated and supplementary intravenous anesthesia was necessary; these three patients had extensive pilonidal sinus tracts. Nausea, sweating and dizziness were noted in five patients and seemed to be correlated with extreme apprehension and emotional instability.

The postoperative complications noted during the period of hospitalization (Table v) were of three types, urinary retention, postspinal headache and discomfort at the site of lumbar puncture. Eleven patients required catheterization but none of these required it on more than one occasion. Four other patients experienced difficulty in voiding but did not need to be catheterized. Four patients suffered typical and severe postspinal headaches. Six other patients complained of mild headaches that were neither incapacitating nor typical. Moderately severe low back pain in relation to the site of lumbar puncture occurred in two instances but in only one was this pain correlated with any difficulty in performing lumbar puncture.

TABLE V
INCIDENCE OF POSTOPERATIVE COMPLICATIONS

Complication	No. of Instances
Catheterization	11
Difficulty in voiding	4
Severe postspinal headache	6
Mild postspinal headache	4
Low back discomfort in relation to site of lumbar tap	3

COMMENTS

One disadvantage encountered with the technic was that of postspinal headache. However, this complication was not foreign to other types of spinal anesthesia; and although this series was limited numerically, the incidence did not appear to be abnormally high. A second disadvantage common to surgical procedures on the anorectal region under any form of anesthesia was a definite incidence of postoperative urinary retention. Whether this complication was due to the anesthesia *per se* or due to the reflex effect produced by the surgical procedure on the autonomic nervous system, was a moot question. A third disadvantage was the reflex abdominal pain when the surgical procedure involved overdilatation of the rectum. The limitation of the extent of anesthesia, one of the method's major advantages, became a disadvantage under certain circumstances.

The level of anesthesia proved to be too limited for some surgical procedures involving extensive pilonidal sinus tracts. A fifth disadvantage was the position utilized in the performance of this technic, a position which was definitely contraindicated in patients with decreased vital capacity due to poor cardiac reserve, chronic pulmonary disease or other conditions impairing pulmonary ventilation.[9]

One of the outstanding advantages of the technic and a source of great appeal to both the anesthetist and the surgeon working on a busy surgical service was the rapidity with which anesthesia could be induced and operation begun. Ability to perform lumbar puncture in the prone-flexed position was easily acquired by a competent anesthetist. The onset of analgesia was prompt and as the patient was already in the optimal position, operation could be undertaken immediately. A second advantage was the prolonged anesthesia that was obtained at the expense of a minimal amount of anesthetic agent. A third advantage was the definite limitation in the extent of anesthesia which ensured the absence of circulatory depression and, since motor power to the lower extremities usually remained intact, permitted immediate ambulation upon completion of the surgical procedure thereby minimizing postoperative vascular and respiratory complications.

SUMMARY

1. Spinal anesthesia utilizing pontocaine in hypobaric solution and administered in the prone-flexed position, is redescribed.

2. This technic was successfully employed in seventy-five instances of surgical procedures involving the anorectal region; in five other instances failures occurred.

3. Complications arising from the use of this type of anesthesia included urinary retention, postspinal headache and discomfort at the site of lumbar puncture.

4. The advantages and disadvantages of the technic are weighed.

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pHISODERM WITH HEXACHLOROPHENE (G-11)*

ITS INTEGRITY AS A SURGICAL SCRUB

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MODERN labor and time-saving devices have stimulated efforts to promote efficiency in all phases of life most particularly in the manpower-saving aspects. The apparent wastefulness of the ten- to twelve-minute soap scrub is detrimentally reflected in the pattern of efficiency in surgical operating room work where delay in any one section of individuals contributing to that work magnifies the lost time as other members of the staff are reduced to idleness. The time factor became extremely important when rigid forty-hour schedules were introduced for all workers except physicians and the destruction of time produced serious economic losses. Realizing that time applied for the benefit of the patient during operations must in no respect be compromised if that time is necessary to obtain sound surgical results, attention must be directed to those time-saving factors which will allow streamlining and at the same time not be deleterious in any manner to the patient's successful surgical progress. The lengthy soap scrub technic is therefore subject to scrutiny and is a candidate for revision.

Previous to the introduction of detergents all methods required ten to twelve minutes of time per individual besides the labor of preparing soap solutions, brushes, containers, basins and antiseptics. Occasional skin reactions prohibited a certain number of operating room personnel from participating in surgical action, often intermittently and rarely permanently. Also, the irritated skins of some participants are questioned from a technical viewpoint due to the fact that bacteria are numerously demonstrated in such skin areas and are therefore dangerous.

Therefore, it appears proper to establish the efficacy of a detergent which will cleanse, lower the bacterial count of the skin, reduce skin irritability, save time and not increase the incidence of wound infection.

pHisoderm plus G-11 (3 per cent)* has been extensively studied in the laboratory and found to meet all of these criteria.[1–10] Several institutions have used this detergent and the staff members are enthusiastic about its safety. Some have used the brush, some have prepared the patient's skin with the detergent, but all have reduced their scrubbing time to three to four minutes.

When experimental evidence was sufficient to predict safety, it occurred to us that the time had arrived for clinical testing; and at Kennedy Veterans Administration Hospital we set up a protocol which appears to be the most rigorous and demanding of all previously reported tests.

PROTOCOL

All features of the surgical management of the patient have remained the same except the actual scrubbing technic of the operating team. The patient's skin is prepared by the usual shave, soap, water, ether, alcohol and merthiolate technic. Antibiotics and other chemotherapeutic agents have been employed with the same frequency and dosage as before the experi-

* Supplied through the courtesy of Winthrop-Stearns Inc.

* From the Surgical Service, Veterans Administration Medical Teaching Group, Kennedy Hospital, Memphis, Tenn. Published with permission of the Chief Medical Director, Dept. of Medicine and Surgery, Veterans Administration, who assumes no responsibility for the opinions expressed or the conclusions drawn by the author.

ment. The same criteria demanded of the condition of the preoperative skin were employed. The only change introduced was the scrubbing technic of the team.

Since July 14, 1948, all operations performed by general surgery, orthopedics, thoracic surgery, urology and neurosurgery were included in the experiment until 1,500 clean cases had been tested.

All wounds have been carefully observed and studied bacteriologically if any degree of infection existed. The report includes all stitch abscesses and gross wound infections. It does not include observations of results obtained by swab cultures around apparently healthy wounds. It is the author's opinion that the demonstration of live bacteria in a clinically non-infected wound is not significant because there has been no interference with healing and no early or late infectious complications.

The observations are made in wounds classified according to ordinary operating room parlance, namely, clean, potentially dirty or dirty. Freeman and Young[11] have used contaminated and infected wounds for the potentially dirty and dirty cases, respectively.

All reddened, indurated areas and seromas and hematomas were cultured. If the cultures were not positive and no subsequent breakdown or stitch abscess occurred, the result was classified as not infected. Mild puffiness or cellulitis appearances are not classified as infected unless subsequent breakdown, abscess or stitch abscess appeared.

Expectant Prescrub State. All scrubbing personnel are requested to maintain clipped fingernails and to exercise normal home cleansing procedures at all times. This request existed for years prior to the start of this experiment.

Scrubbing Technic. Wash hands and arms with pHisoderm and G-11 and water for one minute. Two pushes on container plunger will evict enough material for this purpose (approximately 2 cc.). Rinse with water then cleanse nails with orange stick. Wash hands and arms with same material and water for two minutes then rinse with water. No brushes, no antiseptic washing or soaking are necessary. Actual scrubbing time is four minutes.

Explanation of Clean, Potentially Dirty and Dirty. All operations in which *per primam* healing is expected are considered clean. All operations are considered potentially dirty in which *per primam* healing may be accomplished but in which the likelihood of infection is possible because of varying degrees of contamination. Those conditions considered dirty are those not closed at operation in which *per primam* healing is impossible due to extensive gross infection.

TABLE I
INFECTIONS

Number	Clean	Potentially Dirty	Total
Operations	1,500	430	1,930
Infections	24	17	41
Percentage of infections	1.6	3.95	2.12

ANALYSIS OF RESULTS

During the period of experiment 2,416 operations were performed. There were 1,500 classified as clean, 430 potentially dirty and 486 dirty cases. Obviously all of the dirty operations healed by secondary intention as all were infected. Table 1 contains the results in the cases in which *per primam* healing was expected. An analysis of the infected cases is illustrated in Tables II and III.

COMMENTS

Obviously, defective scrubbing technic is not responsible for all infections; technical errors at the table are responsible for some. For instance, two of the hernioplasty infections were extensive procedures which required scrotal as well as inguinal work; definite breaks were observed. The mild infections seen in removal of ulcerated carcinomas of the face and ear may be due to existing infection in the open lesions.

The two positive cultures in the neurosurgical cases without obvious superficial or deep infection might be errors in the collection of the specimens or in the laboratory. The late infection in the hemispherectomy followed numerous aspirations

table errors are admitted. Also, the results are tabulated from the work of the inexperienced junior resident as well as the experienced senior residents and section chiefs. It is a matter of common knowledge that the experienced operators or teams

TABLE II
INFECTIONS IN CLEAN CASES

Name	Operation	Type	Severity	Organism
M. E.	Craniotomy	Gross	Severe	Hemolytic Staph. aureus
J. S.	Hernioplasty	Stitch	Minimal	Not obtained
J. H.	Hernioplasty	Stitch	Moderate	Not obtained
O. T.	Excision keloids	Stitch	Minimal	Not obtained
L. D.	Carcinoma ear, with skin graft	Stitch	Slight	Not obtained
R. R.	Craniotomy	Late skin infection	Mild	Hemolytic Staph. aureus; P. aeruginosa
O. C.	Hemispherectomy	Gross	Severe	Hemolytic Staph. aureus
B. P.	Tractotomy	Gross	Mild	Hemolytic Staph. aureus
C. B.	Exploratory laparotomy	Stitch	Slight	Staph. aureus
A. McK.	Gastrectomy	Gross	Moderate	Hemolytic Staph. aureus
D. M.	Hernioplasty	Gross	Moderate	B. coli
L. R.	Craniotomy	Gross	Moderate	Hemolytic Staph. aureus
W. L.	Craniotomy	Gross	Severe	A. aerogenes
W. M.	Superficial femoral vein ligation, bilateral	Gross	Moderate	Hemolytic Staph. aureus
K. S.	Burrholes	No clinical infection	.....	A. aerogenes
S. S.	Cranioplasty	No clinical infection	.....	Hemolytic Staph. aureus
H. H.	Cholecystectomy and appendectomy	Partial	Mild	Hemolytic Staph. aureus
R. B.	Plastic flap	Stitch	Mild	Hemolytic Staph. aureus; P. aeruginosa
M. C.	Spine fusion	Stitch	Mild	Hemolytic Staph. aureus
C. O.	Appendectomy	Gross	Moderate	Hemolytic Staph. aureus
A. J.	Gastrojejunostomy; appendectomy	Gross	Moderate	Hemolytic Staph.
H. C.	Open reduction tibia	Stitch	Single	Sterile
A. M.	B/K amputation	Gross	Severe	B. welchii
M. M.	Excision carcinoma face, and graft	Gross	Mild	Not obtained

of the subflap space. One can continue to a point of excusing a great number of these infections from the responsibility of the scrubbing technic. However, technical errors exist as a factor in all wound infections and are so indefinite in their degree of harm that we have discarded them as such. For the purpose of this experiment all infected wounds are included as an indictment of guilt for the scrubbing technic.

Comparative figures are of little value but it appears that any technic which limits the number of infections to 1 to 2 per cent in clean cases cannot be condemned as unsafe. A certain number of technical will work with a lesser number of resulting infected wounds. The results of surgery in the septic age clearly demonstrated this point. It also seems logical to assume that the scrubbing technic is sound if a large number of potentially dirty cases can be performed with an infected rate no higher than 3.95 per cent. Statisticians may require a larger series of cases than we have reported to establish the integrity of this scrubbing technic. But enough experience has been gained by this study so that not a single surgeon employing this technic has expressed apprehension although many were skeptical when the

experiment was launched. No surgeon was commanded to continue this technic if he deemed it unsafe yet no surgeon has returned to the old ten-minute soap scrub.

The technic simplifies the chores of nurses, attendants and physicians. Nurses feel secure that his hands and arms are properly prepared.

It is not possible at this time to comment upon economy. Soap, brushes, containers, alcohol or other antiseptic soaks are items which cost money; pHisoderm and G-11

TABLE III
INFECTIONS IN POTENTIALLY DIRTY CASES

Name	Operation	Type	Severity	Organism
C. L.	Gastrectomy and colon resection	Gross	Severe	B. coli
G. P.	Syme's revision amputation stump	Gross	Moderate	Hemolytic Staph. aureus
B. R.	Choledochojejunostomy	Gross	Severe	B. coli
P. M.	Retropubic prostatectomy	Gross	Moderate	B. proteus; P. aeruginosa; gamma str.
S. B.	Débridement; femoral artery injury	Gross	Severe	Alpha str.; B. proteus; A. aerogenes; E. coli; P. aeruginosa
J. C.	Abdominal evisceration	Stitch	Mild	Not obtained
C. R.	Skin graft, burn, leg	Gross	Mild	Hemolytic Staph. aureus; Beta hemolytic str.
W. H.	Ureter, plastic	Stitch	Mild	Hemolytic Staph. aureus
R. P.	Hemipelvectomy	Partial	Moderate	A. aerogenes; Staph. aureus; alpha str.
J. DiM.	Hernioplasty and orchidectomy	Gross	Moderate	Hemolytic Staph. aureus
D. M.	Exploratory laparotomy; gastrotomy	Gross	Severe	Gamma Str.; Staph. aureus
K. B.	Colectomy	Gross	Severe	Alpha str.; Staph. aureus; coliform intermediate; P. aeruginosa
M. T.	Pilonidal sinus	Gross	Moderate	Staph. aureus
G. W.	Perforated ulcer	Gross	Severe	Beta hemolytic and gamma str.; Staph. aureus; A. aerogenes and P. aeruginosa; Cl. welchii
G. S.	Colostomy	Partial	Mild	B. coli
F. McK.	Perforated ulcer	Stitch	Mild	Staph. aureus
F. G.	Lacerations thigh and calf	Slough of wound	Mild	Sterile

working on a forty-hour-weekly schedule are sorely pressed to prepare materials for the next day's activities. The preparation of sterile materials for the old soap technic required time and effort far above the demands of this simpler technic. The scrub room is tidied and supplied by the attendants with greater dispatch. The physicians' time is saved for more important functions. One of the practical features exists when the supervisory surgeon is called to lend a hand to an inexperienced surgeon. The supervisor can enter the operation without loss of a great amount of time. This phase was of inestimable value in a few instances of extreme emergency. It is possible for the supervisor to be ready for action in a period of three to four minutes and he can

and the dispensers also will require money. We know that approximately 10 gallons of alcohol weekly, a great amount of soap solution and brushes are not needed for this scrubbing technic in our surgical suite. It is theoretically possible for 1,000 individual scrubs to be accomplished from 1 gallon of this semiliquid material. We have learned that the figure is approximately 900 individual scrubs per gallon as there is bound to be some wasting of the material.

No team member has experienced any skin reaction and over 100 different nurses and physicians have participated in this experiment. None of the scrubbing personnel had been known to have suffered significant skin reactions during the old soap method so we are unable to cite

examples of favorable disappearance of skin disturbances by the use of this technic. However, the non-irritability of the skin suggests that avoidance of skin difficulties may be possible with the use of this method.

We have purposely avoided preparation of the patients' skin with this material in order that the scrubbing technic itself could be singly tested. It may eventually prove practical to include this as more evidence of efficacy of the material accumulates.

CONCLUSION

pHisoderm plus hexachlorophene (G-11) 3 per cent has been used as a four-minute, brushless scrub technic in 1,500 clean cases with a wound infection rate of 1.6 per cent and in 430 potentially dirty cases with an infection rate of 3.95 per cent. The results so far suggest that this method is as safe as any previously described scrubbing method. Numerous advantages of the method are briefly discussed.

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A TRANSACROMIAL INCISION FOR EXPOSURE OF THE SHOULDER JOINT*

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THE transacromial incision herein described gives excellent exposure of the shoulder joint. Hence it facilitates operations in this region, particularly those for repair of supraspinatus tendon tears or other ruptures of the tendinous shoulder cuff.

TECHNIC

The skin incision (Fig. 1) begins 2 inches proximal to the distal end of the clavicle (acromioclavicular joint). It continues parallel and posterior to the clavicle, extends over the acromion and ends approximately 1 inch distal to the outer border of the acromion which reaches its maximum width at this point. There is practically no soft tissue between the acromion and the overlying skin.

A knife cut is made through the periosteum in the same direction as the skin incision. This is done to outline the position of the chisel used for the following step, severing the acromion. The chisel used for the osteotomy should be sharp and approximately as wide as the bone.

When the osteotomy has been completed, the severed bones are separated with a strong, self-retaining, sharp-toothed retractor. (Fig. 2.) The fragments are then separated further by hand pressure on the open retractor arms. The exploratory opening is thus widened and a full view of the entire shoulder joint is afforded. Moderate rotation of the patient's arm brings the entire tendinous cuff into sight. Splitting of the deltoid fibers a short distance downward provides ample room for repair of torn tendinous structures. If necessary, the distal portion of the acromial lip may also be removed.

When the operation is completed and the self-retaining retractors are removed, the severed bone fragments close with little pressure. Two or three sutures through the periosteum are sufficient to bring the bone edges into approximation. (Fig. 3.)

COMMENT

Repair of shoulder joint abnormalities through the transacromial incision has given me gratifying results for a number of years. Study of the available literature did not show a procedure similar to the one described here although a number of operative approaches to the shoulder region are on record.

Codman[2] first reported operative intervention for rupture of the supraspinatus tendon as early as 1911. In his interesting, unusual book[3] he mentioned the saber-cut incision. As is well known, this descriptive term represents an anteroposterior cut over the top of the shoulder through the acromion at the acromioclavicular junction.

Codman, however, soon discarded the saber-cut incision for a much simpler one which he called the anterior incision. This extended downward from the anterior acromial edge for a distance of less than 2 inches. The anterior incision permitted (1) fair vision of the underlying bursa and tendinous cuff after separation of the "herring-bone" fibers of the deltoid muscle and (2) visualization of the humeral head following rotation of the humerus. Nevertheless, the anterior incision sometimes made it difficult to repair tendon tears.

In an attempt to correct the disadvantages of the saber-cut incision and the anterior incision a large number of other

* Read at the North Pacific Orthopedic Society meeting October 11, 1947, Portland, Ore.

1

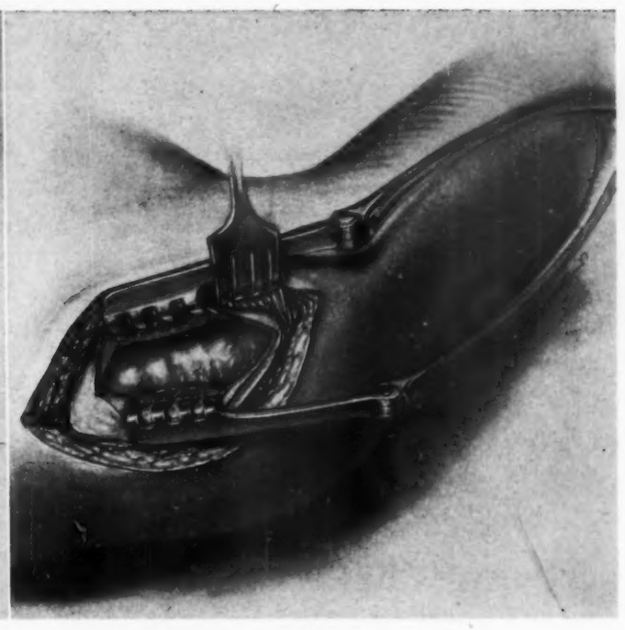

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FIG. 1. Site of acromial osteotomy; skin incision is shown in inset.
FIG. 2. Manner of retracting severed acromion; bursa is visible in wound.

procedures were developed. But, according to McLaughlin,[5] the commonly used approaches to the shoulder joint region are practically always accompanied by one or more of the following handicaps:

(1) inadequate exposure due to obstruction by an intact acromion; (2) a poor cosmetic result due to spreading of a vertical scar; and (3) paralysis and wasting in the anterior portion of the deltoid due to operative interference with axillary nerve branches.

McLaughlin presented a superior procedure based primarily on the saber-cut incision and the posterior approach described by Kocher.[4] In McLaughlin's method, however, the acromion is severed in an anteroposterior direction about midway between the acromioclavicular joint and the lateral border of the acromion. The outer border of the acromion is usually discarded in this procedure; but when it is retained, closure may be accomplished by the use of soft tissue sutures. McLaughlin advised against any attempt at bone suture and said, in his experience union had always taken place without suture of the bone.

Bosworth,[1] whose outstanding contributions to shoulder operations are well known, in 1940 presented twenty-eight cases of incapacitating shoulder lesions which he had radically explored and repaired. Using an inverted L incision he cut the deltoid muscle from its acromioclavicular attachment and retracted backward all of the severed muscle. Bosworth was of the opinion that this division of the deltoid fibers left no permanent clinical defect.

Nicola[6] listed several methods for exposure of the shoulder joint. Among these were the anterior curved approach, which may be enlarged by cutting the deltoid muscle at its origin along the clavicle, and the lateral approach, which begins at the tip of the acromion and extends downward for 2 inches. If the deltoid muscle should be incised more than 2 inches from the tip of the acromion, the anterior circumflex vessels and the anterior branch of the axillary nerve might be injured, Nicola warned.

Nicola also mentioned the anterosuperior posterior incision, extending backward along the edge of the acromion, and the curved anterosuperior posterior approach,

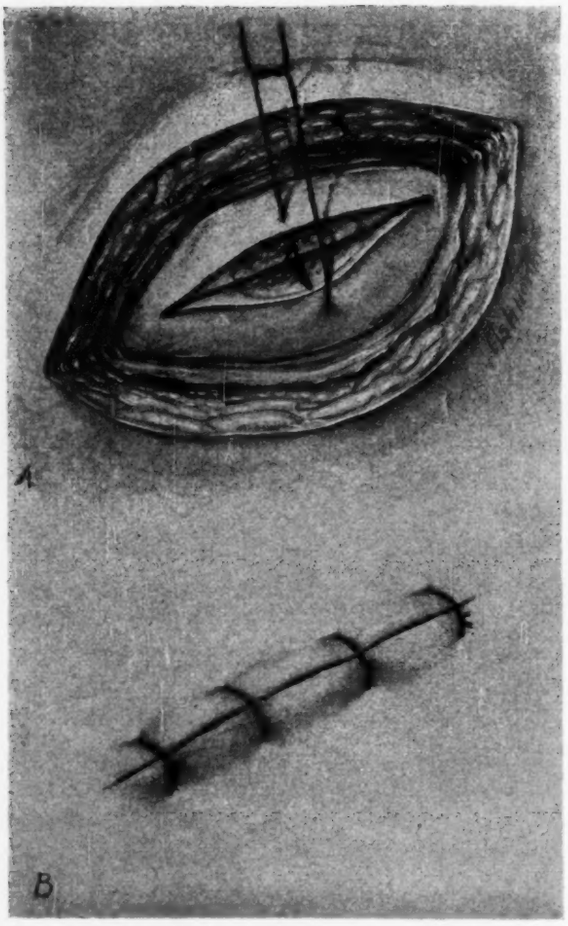

FIG. 3. A, closure of acromion by means of interrupted sutures through the periosteum; B, skin closure.

or Codman's saber-cut. According to Nicola the saber-cut procedure apparently may include severance of a portion or all of the acromial process and may or may not include disarticulation at the acromioclavicular joint.

In my experience repair through the saber-cut incision was frequently followed by non-union of the severed acromion. This was distressing as the severed acromion was clearly visible in roentgenograms. Although the ununited acromion probably caused little disability, the patient, seeing his roentgenogram, often could not escape a feeling of insecurity regarding the results of the operation. Repair through the transacromial incision insures approximation of the severed acromion and subsequent roentgenograms show no bone abnormality.

SUMMARY

1. A simple transacromial operative procedure for exposure of the shoulder joint is presented.

2. Use of this procedure over a number of years proves: it insures adequate exposure of the shoulder joint so that repair of torn tendons is easily carried out; it is safe because no harm can be done to the vessels and nerves of the deltoid muscle; and it gives an excellent cosmetic result.

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THE OXYHEMOGRAPH*

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THE feasibility of using the photoelectric method clinically for determining and continuously recording the oxygen saturation of blood during anesthesia by means of a portable sensitive apparatus was reported by the authors for the first time to the American Surgical Association in 1940.[1] Since that time we have devoted our efforts largely to the development and proving of a portable, stable and reliable amplification and control system that might be used with various types of photocell applicators.

In our work on oxyhemography the chief difficulty encountered has been in instability and inconsistencies seemingly inherent in amplifiers of the conventional D.C. type. Several of our earlier models incorporated these conventional D.C. amplifiers and, although sensitive, were found to be too unreliable for prolonged experimental or clinical application. We were particularly fortunate in obtaining midway in our studies a "contact modulated D.C. amplifier" through Mr. C. F. Kettering which is both stable and sensitive. Further adaptation and the necessary controls were worked out through the collaboration of Mr. F. W. Chapman[2] of the General Motors Research Laboratories.

With the amplifier[3] in its present form the signal from the photocell applicator is converted by the motor driven input breaker to pulsating current of 80 cycles per minute. This 80-cycle current signal is now amplified approximately one million times with the usual resistance-coupled alternating current amplifier. The amplified signal is now rectified by an output breaker to produce an unidirectional output current adequate to operate the ink-writing recording milliammeter. This procedure of amplifying the input signal only after it has been converted to alternating current eliminated the greatest factor of instability in the direct coupled D.C. amplifiers caused by variations in the tube electron streams. Such variations are apt to be large in comparison with the input signal and be insignificant in the amplification factor of the tubes. The contact modulated amplifier avoids this source of instability because the operation is effected by the dynamic amplification factor of the tubes. A second distinct advantage is the breaker system since synchronous rectification in the output circuit allows only those voltages of the same frequency as the input breaker to produce a direct current component in the output circuit. Extraneous fields from other electrical equipment unless identical with the breaker frequency will produce only alternating current components in the output circuit and will not actuate the D.C. recorder.

The operation of the photocell unit depends upon the fact that in the red region of the spectrum reduced hemoglobin absorbs more light than oxyhemoglobin; therefore, when the unit is applied to the tissues, a variation in the red light absorption indicates a change in relative amounts of oxygenated and reduced hemoglobin under observation. Many types of photocell applicators have been devised and may be either of the high-vacuum emissive phototube or the barrier-layer type. We have built and used three of the former type[1] but find the latter type superior largely because the small size and light weight adapts it to use on the ear.

Nicolai[4] was apparently the first to determine oxyhemoglobin photoelectrically but Millikan[5] devised and described a two-

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FIG. 1. The new model oxyhemograph in operation.

color colorimeter a year later. Kramer[6] and Matthes[7] applied the photoelectric method to oxyhemoglobin in tissues in 1934 and 1935 but "volume variables," particularly vasomotor changes resulting from blood pressure fluctuations, were not fully considered until Goldie with his ratiometer and Hemingway and Taylor through their automatic compensation suggested by Millikan[8] attempted to eliminate them as sources of error. Other efforts to correct "volume variables" involve a second spectral region sensitive to total hemoglobin that is near infra red (Squire),[9] blue (Goldie)[10] and green (Matthes[7] and Millikan).[8] There is still controversy as to which method of correction is superior but we have used the Millikan-Rawson cell for the most part. The new model oxyhemograph* (Fig. 1) is so arranged that photocells incorporating either the green or near infra red as the second spectral region may be used.

SUMMARY

A photoelectric apparatus (oxyhemograph)[3] is described which detects anoxemia in early stages not detectable by any other method due to the fact that any change in the light absorption of the hemoglobin produces a signal from the photocell which appears as a recording after a million times' amplification.

The possible applications of this instru-

* Manufactured by Photocon Research Products, 1062 North Allen Avenue, Pasadena 7, California.

ment to physiology and clinical medicine are obviously numerous but we have used it to date principally in the experimental and clinical study of anesthesia, congenital malformations of the heart and cardio-respiratory insufficiencies.

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INTRAMUSCULAR INFUSIONS OF PROTEIN HYDROLYSATE*

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PARENTERAL fluids may be given by the intravenous, subcutaneous, intramuscular, intrasternal and intraperitoneal routes. The intravenous and subcutaneous paths are used most frequently for the infusion of large quantities of isotonic solutions. Little attention is given the intraperitoneal system for adults in this country whereas in the infant this site is employed rather frequently. The intrasternal passage is reserved chiefly for the administration of blood and plasma when the intravenous system is not readily available.

Muscles have long been the path of choice for the injection of small quantities (1 to 10 cc.) of medicaments because of simplicity of injection, the rapidity of absorption and the minimal number of symptoms related to the procedure. Crystalloids, plasma and serum are frequently injected intramuscularly in infants. During the course of the investigation of liquid isosmotic plasma and serum the intramuscular system was found to be a ready route for the infusion of this agent as an adjunctive pathway in the management of shock or for the correction of hypoproteinemia and dehydration.[1]

The protein hydrolysate solution† used in this investigation is a commercially available product prepared from bovine plasma. This preparation has been described in previous reports.[2]

To maintain daily positive nitrogen balance postoperatively by total parenteral alimentation, it is necessary to give 2, 3 or 4,000 cc. of 5 per cent protein hydrolysate solution in the average major surgical procedure.[3] One and a half hours are usually required for the intravenous administration of 1,000 cc. of this protein hydrolysate solution,[4] hence, four to six hours are required for total intravenous alimentation in a twenty-four-hour period. If the intravenous infusion takes three or four hours per 1,000 cc., the patient may be immobilized for as long as twelve hours for total parenteral therapy. The patient, therefore, is in bed the greater part of the day and ambulation is delayed. Likewise, the restricted activity of the arm during intravenous infusions increases the patient's postoperative discomforts. Other handicaps to the use of the intravenous path are the difficulties associated with repeated venipuncture.

We have employed the subfascia lata space and the lateral muscles of the thigh as a route for the rapid infusion of 1,000 cc. of 5 per cent protein hydrolysate solution.[2] The average rate of injection by gravity with this method is 30 to 35 cc. per minute. At this rate 1,000 cc. may be injected in approximately thirty minutes.

Two hundred fifty-four rapid intramuscular infusions into the vastus lateralis muscle of the thighs have been employed in 112 patients without local reactions, irritations or failure of absorption.

Finley and his associates[5] have practiced subfascia lata infusions of 1,000 cc. of isotonic sodium chloride or 5 per cent dextrose solutions. Their report reveals that the average time for delivery of the solution by this route is fifty-six minutes whereas in thirty-three consecutive cases of subcutaneous infusions 167 minutes were

† Supplied by Baxter Laboratories, Inc., Morton Grove, Ill.

* From the Department of Surgery of the George Washington University School of Medicine and the Surgical Department of the Gallinger Municipal Hospital, Washington, D. C.

required for successful injections. One of the early writings on the intramuscular path for the injection of dextrose was that of Nassau in 1926.[6] During the next years only a few reports of the intramuscular injection of dextrose in infants and children appeared in this country and in Germany.[7–10] In 1939 Horsley[11] described the muscular route to be an efficient and simple method for administering dextrose and Ringer's solution.

TECHNIC OF INFUSION

A Y set-up is used for the simultaneous administration of a 5 per cent protein hydrolysate solution to each leg. The bottle of solution is suspended 3 feet above the level of the thigh. Two 15–18 gauge, 2½ inch, long beveled needles are employed. Air is removed from the infusion system by filling the tubes with solution after which the shut-off clamps are closed. The site selected for the injection is the lateral side of the thigh at a point midway between the anterior and posterior surfaces at the junction of the upper and middle thirds. A wheal is made in the skin with 1 per cent novocain at the point of insertion of the needle. Then 5 cc. of novocain are injected into the subcutaneous tissues below the wheal. Fifty cc. of 1 per cent novocain may be placed in the hydrolysate solution to decrease some of the local discomfort. The legs are elevated slightly and are somewhat flexed at the knees so as to relax partially the iliotibial band of the fascia lata. This is best accomplished by placing a pillow under each knee. The needle is held at a 45 degree angle with the long axis of the thigh; the point is directed cephalad and the bevel outward. A sharp, firm thrust at this point puts the needle through the skin and subcutaneous fat. When the needle reaches the iliotibial band, resistance is encountered. An extra thrust is required for penetration through this layer. A sense of "give" is felt when the needle pierces the fascia lata. At this point the hub of the needle is lowered to a 15 degree angle with the long axis of the thigh and is

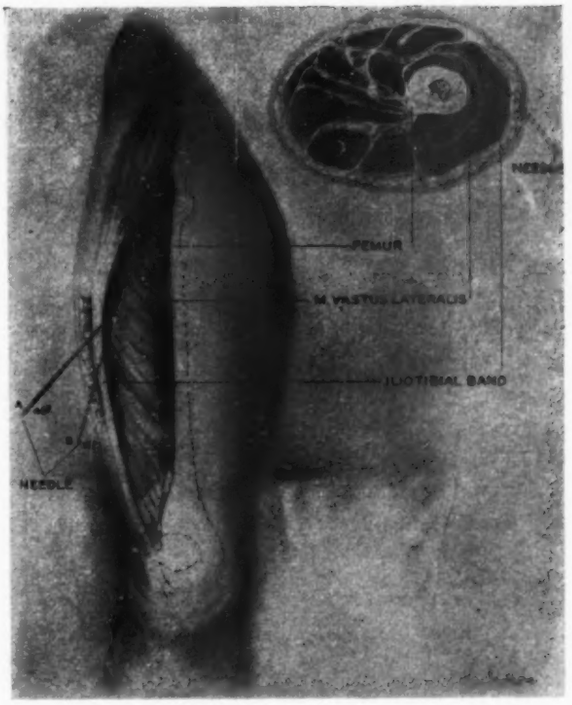

FIG. 1. Shows diagrammatically the method of inserting the needle for intramuscular infusions beneath the fascia lata of the thigh.

advanced for a distance of 1 inch into the substance of the vastus lateralis. It is important to keep the needle at a safe distance from the femur for if the periosteum of the bone is touched by the needle, the patient experiences considerable pain. In the event that the latter occurs inadvertently, it may be used as a guide for the withdrawal of the needle to the selected and correct point in the substance of the muscle. The needle is not taped to the skin because the fascia lata holds it in place. The clamps on the delivery tubes are completely opened after both needles are adequately inserted, thus allowing the solution to "pour in." It is surprising to see how rapidly the first 400 cc. are administered—four to five minutes. With the rapid infusion of the first few hundred cubic centimeters the patient notices a smarting, stretching sensation and a feeling of fullness in the thighs at the site of the injection. The next 300 cc. require about eight to ten minutes. A bulging in the lateral compartment of the thigh is noted with the injection of 500 cc. The original

manifestations described before are replaced by the symptom of a heavy feeling in the legs. The last 300 cc. usually consume fifteen to twenty minutes. This slowed rate of infusion is due to the increased tension produced by the large amount of solution present in the confined space of the lateral compartment of the thigh. It may be said that the rate of administration is inversely proportional to the tension in the compartment produced by the quantity of injected fluid. (Fig. 1.)

TABLE I

BLOOD AMINO ACID AND UREA NITROGEN CHANGES AFTER RAPID INJECTION IN BOTH LEGS OF 1000 CC. OF PROTEIN HYDROLYSATE SOLUTION CONTAINING 50 GM. HYDROLYSATE IN THIRTY MINUTES

Constituent Analyzed	Before Injection	Half-hour-end of Injection of 1,000 cc.	One Hour Later	Two Hours Later	Three Hours Later	After Twenty-four Hours
Amino acid nitrogen.....	6.2*	7.6	7.4	7.0	6.5	6.2
mg. per 100 cc. plasma.....	5.8–6.6†	6.8–7.8	7.2–7.7	6.8–7.4	5.9–7.0	5.7–6.5
Urea nitrogen mg. N.....	12.4	12.8	12.6	12.8	11.8	12.3
per 100 cc. plasma.....	10.0–13.9†	11.0–14.0	10.5–13.6	10.8–14.2	10.9–13.0	9.6–14.2

* Average values
† Range of values
Number of cases: 10
(From WEINSTEIN, J. J. Intravenous, subcutaneous and rapid intramuscular infusions of "protein hydrolysate." *Surg., Gynec. & Obst.*, 87: 12, 1948.)

TOLERANCE AND ABSORPTION

The patients receiving protein hydrolysate intramuscularly were the postoperative surgical patients requiring parenteral fluids and protein nutrition, or the preoperative patient with a history of malnutrition, weight loss and measurable hypoproteinemia who could not take adequate amounts of protein orally (viz. pyloric obstruction, ulcerative colitis). Thus, rather ill surgical patients were used in this investigation. The largest amount of protein hydrolysate administered by the muscular path to one patient was 28,000 cc. in a fourteen-day period.

A review of the effect on blood amino acid nitrogen and urea after the intramuscular injection of 1,000 cc. of protein hydrolysate solution in thirty minutes indicates an average rise of amino acids of 1.06 mg. per 100 cc. of plasma above normal at the end of the injection, a 1.2 mg. elevation one hour later, a sustained elevation of 0.8 mg. two hours later and a retained rise of 0.3 mg. three hours after completion of the infusion. The urea figures do not indicate significant changes for the same periods. Table 1 gives the average values and the range of change in blood amino acids and blood urea for ten patients who received 1,000 cc. of protein hydrolysate solution rapidly intramuscularly. The amino acid changes expressed in terms of per cent rise above normal and per cent difference for each period as compared to the maximal rise seen at the end of the injection is described in Table II.

TABLE II

CHANGES IN AMINO ACIDS AS PER CENT ABOVE NORMAL AND PERCENTAGE DIFFERENCE BETWEEN EACH PERIOD AS COMPARED TO MAXIMUM LEVEL

	End of Infusion	One Hour Later	Two Hours Later	Three Hours Later
Percentage rise above normal.....	15.4*	19.3*	13.0*	4.0*
Difference in percentage change.....	15.4*	3.9*	−6.3	−8.2

* Average values.
Number of cases: 10.

Further evidence for the rapid absorption of protein hydrolysate given intramuscularly is observed in an analysis of the absorption and excretion rate of 1 cc. of phenolsulfonphthalein dye that is added to the 1,000 cc. solution to be injected. Analysis of this experiment shows 46 per cent of normal absorption and excretion of the dye one hour after the end of the infusion, 77 per cent of normal absorption at the two-hour postinfusion period and 93 per cent of normal absorption and excretion of the dye in the hydrolysate three hours after the injection. A total absorption and excretion of 96 per cent of dye was found four hours following the injection.[2]

Leg measurements performed concomitantly with the study of phenolsulfonphthalein absorption and excretion reveal a maximum increase in diameter of the thigh at the end of the infusion (2.75 inches above preinfusion level) and a return to normal size within three hours following the injection.

Figure 2 gives the per cent excretion of phenolsulfonphthalein in the control test, the per cent excretion of the dye when 1 cc. is added to the 1,000 cc. of protein hydrolysate infused intramuscularly in thirty minutes, the per cent excretion of control and the average changes in leg measurements.

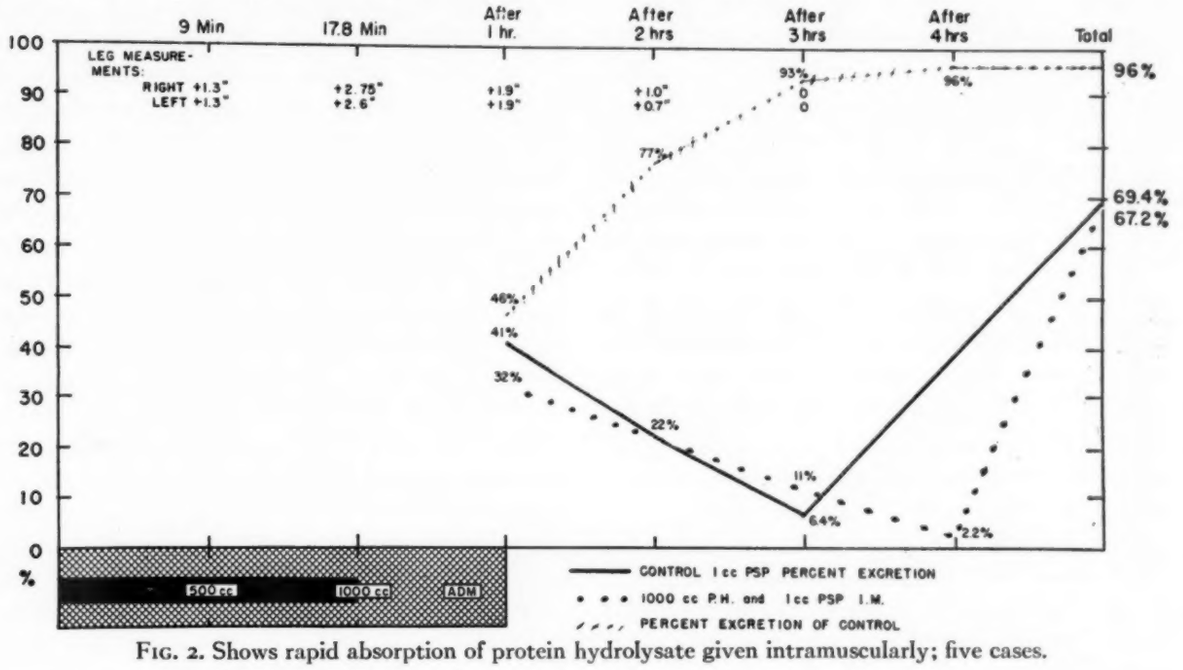

FIG. 2. Shows rapid absorption of protein hydrolysate given intramuscularly; five cases.

SUMMARY AND DISCUSSION

Medical and surgical patients may have a deranged protein balance because of limited or inadequate intake, loss through abnormal routes, excessive catabolism, faulty digestion and absorption, and abnormal utilization and fabrication of proteins. When the oral route is available and efficient, this is certainly the path of choice for protein feeding. However, there are numerous instances wherein the oral route is not available or it may be contraindicated because of disease. Therefore, the parenteral channel must be resorted to for maintainence of daily nitrogen nutrition to help build up depleted reserves and to correct hypoproteinemia. The intravenous route is used most frequently but has its disadvantages. Some of these are as follows: accessible veins may not be available; the size of the veins may make repeated infusions impractical and difficult; trained personnel may be unavailable for efficient intravenous injections; and repeated venipunctures unless performed with extreme

care produce fibrosis and thrombosis which, in turn, limit the life of the accessible and usable veins.

A plausible technic for the administration of protein hydrolysate solutions is a method which is simple to perform, does not require great skill, insures absorption, avoids long periods of immobilization, eliminates the discomforts associated with fixation of the arm for hours and decreases the total incidence of local and general reactions. Rapid intramuscular injections of protein hydrolysate (prepared from bovine plasma) into the lateral surface of the thighs fulfill the aforementioned criteria.

Analysis of this study shows that there is maximum absorption the first two to three hours after the end of the injection in thirty minutes of 1,000 cc. of 5 per cent protein hydrolysate solution. Hence, intramuscular infusions of 1,000 cc. each may be performed and repeated every six hours with maximum safety and efficiency.

The blood amino acid levels attained after rapid intramuscular infusion of 1,000 cc. in a half hour indicate that the maximal levels reached by this route are only one-half that seen with the intravenous infusion of similar amounts in one hour. Therefore, the likelihood of systemic reaction is considerably reduced.

Many institutions are afflicted with a shortage of personnel. Hence, the intramuscular method of infusion could be safely and efficiently executed by the nursing staff so as to relieve the active internes or residents for more pressing duties. It is my opinion that a more efficient service for parenteral therapy could be maintained in hospitals by the use of a trained team of physicians or technicians rather than the usual practice of having each intern or resident on the case do the injection or transfusions. Proof of this is seen in operating rooms where the anesthetists are solely responsible for maintenance of fluid and blood volume during surgery. Also, throughout the years of managing a blood bank in a hospital I have seen greater efficiency in phlebotomy when a trained group of technicians or nurses are used in preference to the floating intern or resident.

In the course of management of postoperative surgical patients it has been found necessary to give 3,000 or 4,000 cc. of protein hydrolysate solution per day to each patient for positive nitrogen balance with total parenteral alimentation. If the intravenous system is employed, at least four and a half to six hours of immobilization are required to administer this quantity. In the event of slower rates of infusion than the usual 10 to 15 cc. per minute the patient may be immobilized for twelve hours or more for the successful infusion of 3,000 cc. of fluid. This is indeed a handicap to the postoperative patient and decreases the likelihood of early ambulation and its concomitant benefits. Therefore, we have added the rapid intramuscular method of infusion to our program of postoperative parenteral nutrition. A simple program that will maintain positive nitrogen balance postoperatively in the greater number of cases is as follows: 1,000 cc. of protein hydrolysate with dextrose in the morning intravenously in one and a half hours, 1,000 cc. of protein hydrolysate, plain, intramuscularly in the afternoon in thirty minutes and 1,000 cc. of protein hydrolysate with dextrose intravenously in the evening. Thus only three and a half to four hours of the day are used for infusions and the patient is free for active motion in the remaining periods. Additional infusions may be given intramuscularly or intravenously.

Total parenteral alimentation is performed not only in surgical patients but also in medical patients with acute inflammatory lesions of the gastrointestinal tract, viz., regional ileitis, ulcerative colitis or obstructive lesions of the upper gastrointestinal tract being prepared for surgical intervention. A recent experience with a patient who was operated upon for acute appendicitis but in whom an extensive, acute regional ileitis with perforation was found, bears out the advantage of using

the intramuscular route for infusions in patients on total parenteral alimentation. Few accessible veins in the arms, hands or feet could be found in the patient; in fact the only readily available vein was the internal saphenous near the ankle. Because of the inherent technical difficulties of maintaining catheterization of a vein for continuous intravenous administration for the planned fourteen-day period, the intramuscular route was selected. One thousand cubic centimeters of protein hydrolysate were infused by the intramuscular path twice a day for fourteen days. Repeated blood studies revealed no significant drop in the total protein or albumin content during this two-week period. However, an approximate weight loss of 11 pounds was seen after ambulation.

CONCLUSIONS

1. A protein hydrolysate solution containing 50 Gm. of protein in 1,000 cc. of distilled water can be given intramuscularly into both thighs simultaneously at a rate of 25 to 30 cc. per minute in 1,000 cc. doses.

2. The injections are made into the lateral side of each leg simultaneously with a 2½ inch 15–18 gauge needle at a point midway between the anterior and posterior surface of the thigh and at the junction of the upper with the middle thirds. The needle lies well in the substance of the vastus lateralis.

3. The local discomfort associated with these infusions is minimal and no local systemic reactions have been seen in 254 consecutive injections.

4. The time required for absorption as shown by blood amino acid levels, by leg measurements and by the excretion of phenolsulphonpthalein dye, which is used as an indicator in the solution, is three hours.

5. In view of the absorption rate with intramuscular infusions of 1,000 cc. of protein hydrolysate it is suggested that each dose of 1,000 cc. be given at six-hour intervals.

6. The intramuscular route is a safe pathway for the rapid infusion of large amounts of 5 per cent protein hydrolysate solution.

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ETIOLOGY OF PRIMARY VARICOSE VEINS*

HISTOLOGIC STUDY OF ONE HUNDRED SAPHENOFEMORAL JUNCTIONS

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MUCH confusion and wide divergence of views exist in the voluminous literature on the etiology of primary varicose veins in the lower extremities. The various theories, however, can be placed in two main groups, one stressing mechanical or hemodynamic factors and the other stressing inherent structural weakness of the veins themselves. The mechanical or aggravating factors are well known but the exact nature of the hereditary defect or inherent venous weakness has remained obscure. Variations in pattern and size of the same veins and their tributaries in different individuals as well as the marked variations in valves with respect to number, position, efficacy and the intervals between them present obstacles in interpretation not easily overcome. The saphenofemoral junction was chosen for histologic study because it is a constant structure anatomically; because the valve guarding its orifice is the most constant one in the entire saphenous system; and because the great saphenous at this area is a perforator vein at a common site of retrograde blood flow in varicose states.

MATERIAL AND METHOD

One hundred junctions were obtained at necropsy from sixty-nine consecutive cadavers. Five specimens were from three infants, four specimens from two children and ninety-one specimens from sixty-four adults. Eight specimens of the latter were obtained from six cadavers in which it was possible to make a diagnosis of varicose veins at the time of autopsy.

Each saphenofemoral junction was removed by bisecting the inguinal ligament, retracting the overlying skin and exposing the veins *in situ* without traction. The saphenous vein was severed transversely 1 cm. below the junction; the femoral vein was cut across an equal distance above and below the junction and the entire segment was removed *in toto*. The lumen of each vein was loosely and carefully filled with absorbent cotton from below so as not to exert pressure upon the valve leaflets and the whole specimen was fixed in 10 per cent formalin. After fixation a longitudinal section was made through both the femoral and saphenous veins. One-half of the bisected junction was discarded whereas the other half was embedded in paraffin. Triplicate histologic sections were stained with hematoxylin and eosin, with Weigert's elastic tissue stain and with Weigert's elastic tissue stain using picric acid and acid fuchsin as counterstain.

RESULTS

Of the entire group of 100 there were only sixteen patients in whom the sinus wall of the saphenous vein opposite the valve was histologically normal; there were eighty-four in whom the sinus showed a defect in the wall that is described hereafter. This defect was present in each of the specimens from cadavers with varicose veins, in each of the four specimens from the two children, in four of five specimens from the three infants and in sixty-eight specimens from adults without apparent varicosities.

In all sections the wall of the saphenous vein both above and below the sinus and valve was normal. (Figs. 1 and 2.) The tunica intima consisted of an inner lining

* From the Samuel D. Gross Surgical Division and Clinical Laboratories of the Jefferson Medical College Hospital, Philadelphia, Pa. Presented before the Philadelphia Academy of Surgery, February 3, 1947.

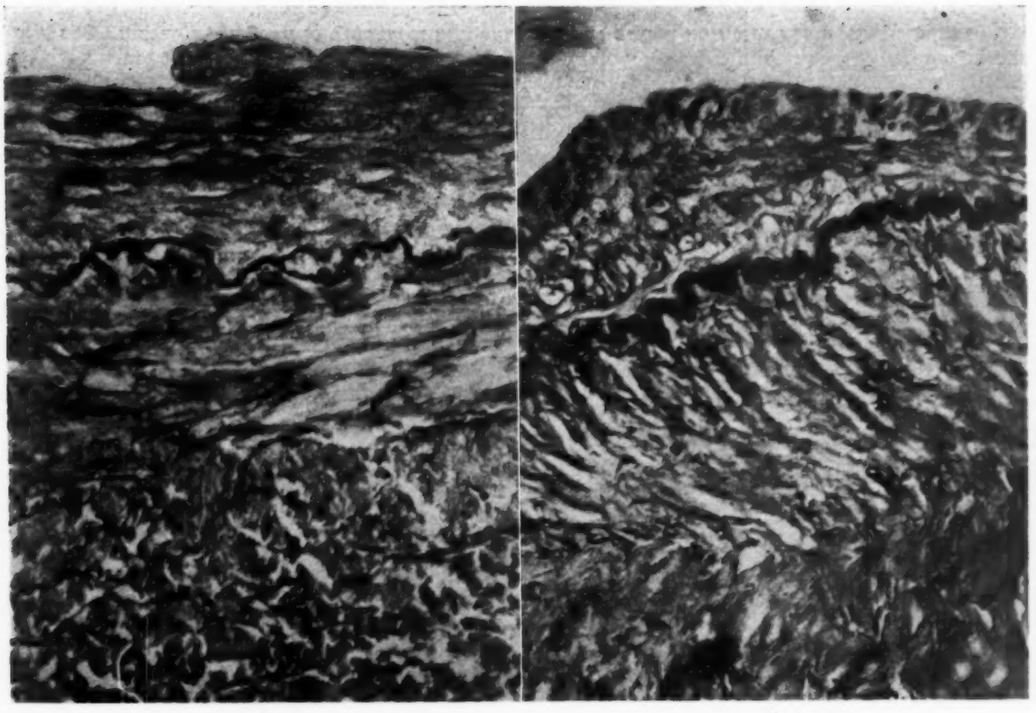

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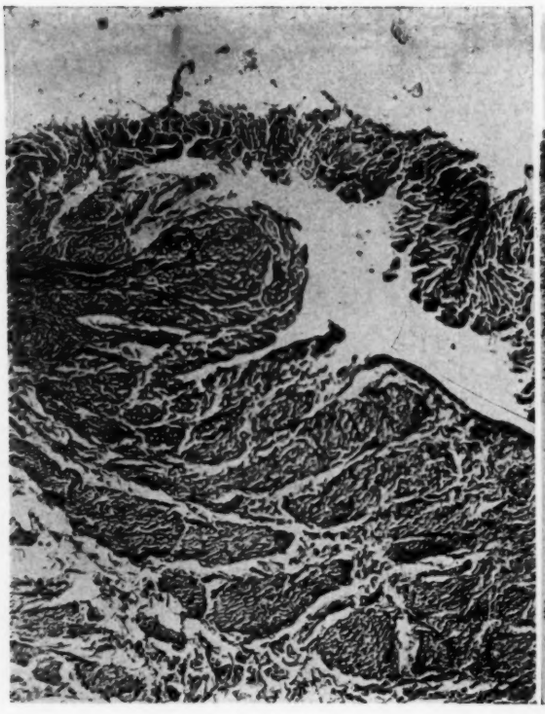
FIG. 1. Section of a normal saphenous vein above the valve showing a thick tunica intima, a well defined internal elastic membrane and a portion of the tunica media; the latter discloses numerous muscle bundles interspersed with a few collagen bundles and elastic fibers. (Elastic tissue stain × 200.)

FIG. 2. Section of a normal saphenous vein distal to the valve showing a structure similar to that illustrated in Fig. 1. (Elastic tissue stain × 200.)

of endothelial cells, an intermediate layer of connective tissue and an outer layer of thin elastic fibers which frequently coalesced to form a definite internal elastic membrane. The tunica media was composed of a broad layer of smooth muscle bundles between which were collagen bundles and coarse elastic fibers. The tunica adventitia was usually as thick as the media and was composed of dense acellular collagenous bundles interspersed with numerous thick elastic fibers. The valve leaflets were also normal. They consisted of a thin strand of connective tissue which was covered on its distal surface with a broad band of elastic fibers. (Figs. 3 and 4.) At the base of the valve the latter was continuous with the internal elastic membrane of the tunica intima. The proximal surface of the valve was devoid of elastic fibers.

In normal saphenofemoral junctions the sinus wall was only slightly thinner than the wall of the adjacent portions of the saphenous vein. (Fig. 3.) Although somewhat less distinct all coats of the vessel wall were identifiable. The internal elastic membrane was less conspicuous but was nevertheless present and was directly continuous with that covering the more proximal segment of the saphenous vein.

In defective saphenofemoral junctions the sinus wall was thinner from one-half to one-fifth of the normal thickness. (Fig. 4.) The tunica intima contained no internal elastic membrane although in a few cases scattered, short, ill-defined elastic fibers were present. (Fig. 5.) The tunica media, when recognizable as such, disclosed only a few small bundles of smooth muscle, a more abundant amount of collagenous connective tissue and only scattered elastic bundles. Not infrequently, particularly at the base of the valve leaflets, there were no muscle bundles whatsoever and the few collagen bundles that formed this coat



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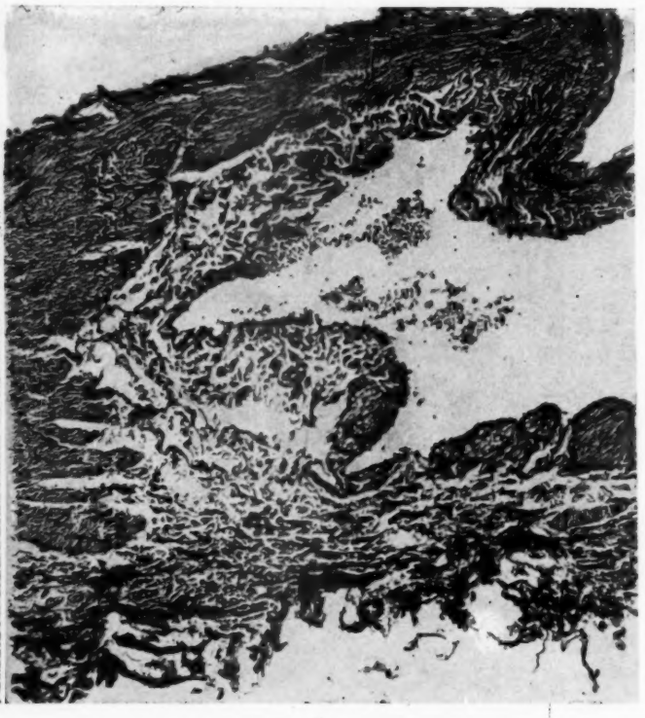

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FIG. 3. Normal saphenous sinus wall; the tunica intima contains elastic fibers. The tunica media is thick and composed of well developed muscle bundles, collagen bundles and elastic fibers; note the elastic fibers covering the distal surface of a valve leaflet. (Elastic tissue stain × 75.)

FIG. 4. Defective saphenous sinus wall showing a thinning of the coats, an absence of an internal elastic lamina of the tunica intima and of muscle bundles of the tunica media and a paucity of elastic fibers in the middle coat. (Elastic tissue stain × 75.)

were intimately blended with the tunica adventitia. The latter varied in thickness from that covering other portions of the vein to a layer about one-third or one-quarter that of normal. The proportion of collagen bundles to elastic fibers did not differ from that in other portions of the saphenous vein.

Nine femoral veins were sectioned in the valve area located just above the entrance of the saphenous vein for comparison with the saphenous veins. In each the sinus wall was similar to that found in the sixteen normal saphenofemoral junctions. (Fig. 6.) None showed the defects encountered in the remaining eighty-four specimens.

COMMENTS

Thinness of the saphenous vein wall at the sinus area of the valve was of such frequent occurrence in this series as to suggest its normalcy in the majority of cases. That an attenuation in this area of the wall is of normal congenital and developmental origin was first shown by Kampmeier and Birch[1] who studied the embryologic development of the venous valves in the human fetus and newborn. They observed that in the final stage of formation of the valves and their sinuses there was a bulging of the vein wall sequential to a local retardation in growth and a deficiency in circular muscle development of the tunica media. At such points in the newborn the latter was only one-fourth to one-fifth as wide as elsewhere in the vein. The disparity in the thickness of the wall and in the disposition of the muscle fibers was interpreted as having importance in the functioning of the valve.

Granted that a certain degree of thinning of the venous wall at the sinus area is normal, it must be conceded that there are limits beyond which the condition must be regarded as definitely abnormal. Loss of circular muscle fibers is the rule but some

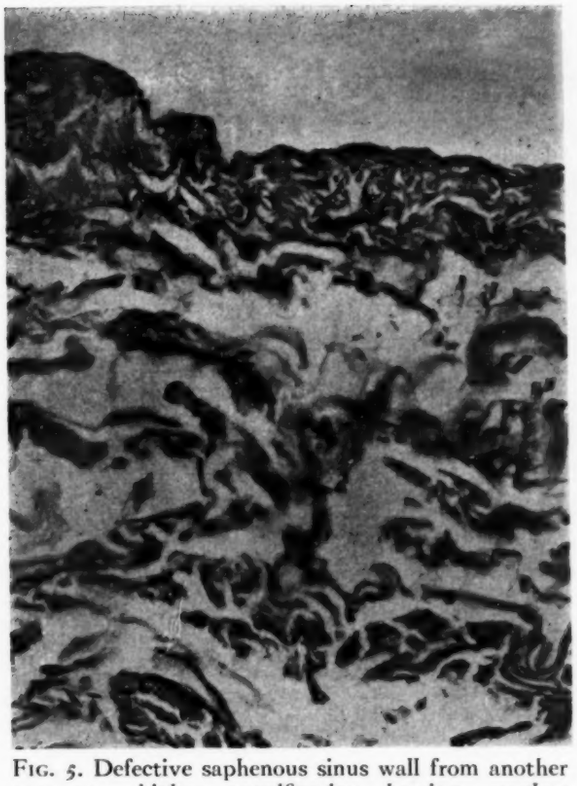

FIG. 5. Defective saphenous sinus wall from another case at a higher magnification showing complete absence of muscle bundles and a paucity of elastic fibers. (Elastic tissue stain × 200.)

FIG. 6. Normal femoral sinus wall showing a structure similar to that of a normal saphenous sinus wall illustrated in Figure 3. (Elastic tissue stain × 37.5.)

muscle, usually longitudinal fibers, as well as some form of elastica should be present. According to the studies of Clark[2] smooth muscle in a state of tone should form a physiologic first line of defense against distention of the vessel wall. When this resistance has been completely overcome, the fibro-elastic components are forced to take over. It is thus likely that a sinus wall in which there is complete absence of muscle as well as internal elastic membrane will eventually dilate under the repeated stress of internal mechanical forces.

The most formidable of the hemodynamic or aggravating forces is probably the hydrostatic pressure which is considerably higher in humans than in lower animals because of the erect posture. The importance of the role played by this pressure is emphasized by the fact that varicose veins as seen in man do not occur in quadrupeds. Furthermore, as pointed out by Eger and Casper,[3] in those individuals with absence of valves in the external iliac and femoral veins above the orifice of the great saphenous an even greater burden of support is imposed on the valves of the latter vessel. Increase in intra-abdominal tension in pregnancy, pelvic tumors, heavy muscular work with straining at lifting and repeated straining at stool exert pressure on the pelvic veins, thus retarding the venous return. Finally, occupations requiring standing for long periods favor stasis by curtailing the massaging action of muscular contractions on the deep veins, thus retarding the emptying of the superficial veins.

In predisposed individuals the actual varices rarely manifest themselves before adolescence for the following reasons: The hydrostatic pressure reaches its maximum only when full height has been attained; the elasticity of skin and veins is greatest during this period and the diameter of veins relatively smaller; muscular movements are more active and more constant, thus increasing the return flow of blood and encouraging drainage of the superficial veins; the veins are still growing in length; and occupations involving protracted periods of standing are seldom required.

It is significant that in this study the valve leaflets themselves were histologically normal in all instances including the cadavers with varicose veins. Although congenital absence of valves, degeneration with increasing age and destruction by thrombophlebitis have been reported, we do not believe that such valve defects *per se* constitute a common cause for the development of varicose veins. Indeed, it is much more probable that in the usual case the valvular insufficiency is purely functional and secondary to dilatation of the vein wall.

With regard to the hereditary tendency toward the development of varicose veins there is little dispute. Figures indicating this influence vary between 40 per cent[4] and 65 per cent.[5] If blood relatives other than siblings or parents are included, the percentage is approximately 90 per cent. It has been observed that not only may several members of the same family in succeeding generations have varicosities but also the pattern of the involved veins may be similar in all. It is reasonable to suspect that such defects might be on an embryologic or developmental basis. For this reason we sought a muscular defect in the tunica media at the bifurcation angle of the saphenofemoral junction. Such a defect was described by Forbus[6] for arteries in the circle of Willis bearing congenital berry aneurysms and was recently encountered by us in a case of aneurysm of the inferior pancreaticoduodenal artery.[7] In each instance, however, the muscle coat of the saphenous vein showed no evidence of faulty fusion with the muscle coat of the femoral at the junction of the two veins. The structural defect was found instead at the sinus wall of the vein.

SUMMARY AND CONCLUSIONS

1. Absence of smooth muscle and internal elastic membrane in the sinus wall of the great saphenous vein in the valve area at the saphenofemoral junction is fairly common.

2. This defect may be congenital and possibly hereditary.

3. It is possible that such defects in other perforator veins would predispose to dilatation under the constant stress of hemodynamic forces, rendering the corresponding valves functionally incompetent and permitting retrograde flow of blood from the deep into the superficial veins.

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EFFECTS OF URONIC ACIDS, PECTINS AND PECTINATES ON THE ENTERIC FLORA, ALONE AND IN COMBINATION WITH ANTIBIOTICS*

I. *IN VITRO* STUDIES

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RECENT clinical and experimental work by Mast and Harrison[1] has indicated that various pectins, pectinates and uronic acids when given alone by mouth possess no potent *in vivo* antibacterial properties but become extremely powerful bactericidal agents in the gastrointestinal tract when given together with oral streptomycin. In combination these drugs far surpassed the ability of streptomycin alone to eliminate intestinal organisms. There were important advantages possessed by these combinations regarding subsequent development of streptomycin resistance by the enteric flora. They found it possible to sterilize the feces completely in several patients and to eliminate all but a few spore-forming anaerobic organisms in others. This work has since been confirmed by Pulaski[2] and further by our own clinical studies now in progress.

The ability of pectin compounds to give subjective relief in cases of various diarrheas has been known for many years and dates from the discovery of the beneficial effect of scraped apple diets in these conditions. This effect of pectins has been attributed to their ability to adsorb toxins and enmesh offending organisms, to become hydrophyllic to an extreme degree, thus removing excess water from the intestinal tract, and to their mechanical ability of coating the intestinal wall with a smooth, protective and soothing carbohydrate film.

Laboratory studies investigating the antibacterial properties of pectins have revealed several interesting findings. Many investigators have held that the pectins alone possess no antibacterial properties except by virtue of their low pH values in solution. However, when pectins exist in the form of metallic pectinates, certain of the salts become bactericidal both *in vivo* and *in vitro* and such activity has been distinct from pH effect. The pectinate most investigated has been the nickel salt. It has been shown by Haynes and associates[3] and by Myers and Rouse[4] that the presence of small amounts of nickel incorporated into the molecule as nickel pectinate rendered that salt definitely bactericidal. It has also been shown by Myers and Rouse[4] and by Arnold[5] that silver pectinate is even more efficient in this respect and that copper pectinate has a definite toxic action on the Staphylococcus aureus. The amounts of metal in those dry preparations are extremely small and vary from 0.3 to 0.5 per cent. Of this material, solutions of only 1 to 5 per cent have exhibited their strongly antibiotic properties. It is evident that the bactericidal properties of these pectinates cannot be accounted for only on the basis of their slight metal content.

The composition of the many pectins, while varying in some respects, consists of several essential components among which are galactose, arabinose, acetic acid and galacturonic acid.[4,6] Of these, galacturonic acid makes up 87 to 95 per cent of the total and is obtained in the hydrolysis of the complete pectin. There is a possibility

* Studies, observations and reports from the Department of Dermatology of The Barnard Free Skin and Cancer Hospital and the School of Medicine, Washington University, St. Louis, Mo. This work was accomplished under a grant from the Commercial Solvents Corporation.

that the antibacterial properties of the pectin, when given orally with streptomycin, are due to the breakdown of the pectin molecule with liberation of large amounts of galacturonic acid as the active antibacterial principle.[1] When given orally with streptomycin this acid seems to be even more effective than pectin. A closely related compound, glucuronic acid, is similarly effective. Following the administration of these materials it has been found that the portion of the intestinal canal in which feces are sterile, or nearly so, is the lower colon. It has not yet been determined whether or not this is true for the small bowel. It is in the colon also that Werch and his associates[7] found the breakdown of pectins to occur. They discovered the decomposition of the pectin to be due to the action of several organisms all of which were non-pathogenic and which constituted part of the normal enteric flora. They also found that both pectins and galacturonic acid were very slightly absorbed and, in the main, were excreted unchanged in the feces.[8] They acknowledged that both pectins and galacturonic acid were lethel to pathogenic organisms under certain conditions but thought that this was a pH function.

The purpose of this study has been twofold: first, to ascertain whether or not various pectins and uronic acids possess significant antibacterial properties themselves and, second, whether those compounds in combination with various antibiotics show the synergism *in vitro* that exists in the gastrointestinal canal.

TABLE I

PH OF PECTIN, PECTINATE AND URONIC ACID AQUEOUS AND BROTH SOLUTIONS

	Concentration of Solutions															
	¼%		½%		1%		1½%		2%		3%		4%		5%	
	Broth	Aqueous	Broth	Aqueous	Broth	Aqueous	Broth	Aqueous	Broth	Aqueous	Broth	Aqueous	Broth	Aqueous	Broth	Aqueous
7% methoxyl pectinic acid	...	...	6.0	5.0	5.5	4.4	5.0	4.0	4.5	3.8	4.2	3.6	3.9	3.4	3.7	3.2
5% methoxyl pectinic acid	...	...	6.5	5.3	5.9	4.7	5.5	4.2	5.3	3.9	5.0	3.7	4.7	3.4	4.5	3.2
Bismuth pectinate	...	...	6.5	4.2	6.0	3.7	5.6	3.5	5.1	3.4	4.5	3.2	4.0	3.1	3.5	2.8
Aluminum pectinate	...	...	7.0	4.2	6.3	3.8	6.0	3.5	5.5	3.4	4.4	3.2	4.0	3.0	3.7	2.8
Silver pectinate	...	...	5.5	4.0	5.0	3.8	4.8	3.7	4.5	3.6	4.0	3.5	3.7	3.2	3.3	3.0
Galacturonic acid hydrate	5.0	3.6	4.5	3.4	3.7	3.2	3.4	2.8	3.0	2.5	2.7	2.2	2.4	1.8	1.9	1.5
Glucuronolactone	6.8	5.0	6.5	4.7	6.2	4.5	5.8	4.2	5.5	3.9	4.8	3.5	4.4	3.3	4.0	3.0

Throughout the following experiments the test organisms employed were beta-hemolytic streptococci, Staph. aureus, Pseudomonas aerugenosa and Escherichia coli. All were freshly isolated from patients. The culture medium throughout was tryptose broth (Difco). The test substances investigated included 7 per cent methoxyl pectinic acid, 5 per cent methoxyl pectinic acid, bismuth pectinate, aluminum pectinate, silver pectinate, galacturonic acid hydrate and glucuronolactone. The latter compound is a stable inner anhydride which becomes glucuronic acid in solution. These solutions were all prepared by stirring the powder into cold water and then dissolving it with as little heat as possible. Sterilization was accomplished at 15 pounds' pressure for twenty minutes. Following inoculation with the test organisms the solutions were incubated for twenty-four to forty-eight hours at 37°c. After that time the cultures were examined for macroscopic growth. Cultures showing no apparent growth were

subcultured on tryptose broth to check their apparent sterility further.

The pH of various aqueous and broth concentrations of the test materials is shown in Table I.

Because it was found that the beta-hemolytic streptococcus would not grow even in very small concentrations of the test compounds, it was not included in many of the following experiments. The remaining three organisms, however, were incubated in tryptose broth adjusted to diminishing pH values by the addition of hydrochloric acid as is shown in Table II. It was thus shown that all tested organisms would grow well down to and including a pH of 4.5. At a pH of 4.0 growth was still visible but was less than in the higher pH range. (Table II.)

TABLE II
GROWTH OF TEST ORGANISMS IN TRYPTOSE BROTH AT ADJUSTED pH LEVELS

pH	Organism		
	Ps. aerugenosa	S. aureus	E. coli
7.0	+	+	+
6.0	+	+	+
5.5	+	+	+
5.0	+	+	+
4.5	+	+	+
4.0	+ growth diminished at pH 4.0	+ growth diminished at pH 4.0	+ growth diminished at pH 4.0

With this information at hand experiments were then undertaken to determine the concentrations of solutions of pectins, pectinates and uronic acids which might prove bactericidal. These data are presented in Tables III, IV and V.

It was therefore found that unbuffered solutions of all the tested compounds possessed antibacterial properties and, moreover, that such properties were not dependent on pH as bactericidal action was apparent at pH ranges well above that lethel to the test organisms. The pectinic acids were the least potent in that respect; the pectinates with the exception of the silver salt were intermediate and the most effective were silver pectinate, the galacturonic and glucuronic acids. Although our work has included only 5 and 7 per cent methoxyl content pectins, it would seem that the pectin with the highest methoxyl

TABLE III
GROWTH OF TEST ORGANISMS IN DILUTIONS OF PECTINS AND PECTINATES IN TRYPTOSE BROTH

Preparation	Concentration of Solution (Per cent)	Organisms		
		Ps. aerugenosa	S. aureus	E. coli
7% methoxyl pectinic acid	5	−	−	−
	4	−	−	−
	3	−	−	−
	2½	−	+	+
	2	+	+	+
	1½	+	+	+
	1	+	+	+
	½	+	+	+
5% methoxyl pectinic acid	5	−	+	+
	4	+	+	+
	3	+	+	+
	2½	+	+	+
	2	+	+	+
	1½	+	+	+
	1	+	+	+
	½	+	+	+
Bismuth pectinate	5	−	−	−
	4	−	−	−
	3	−	+	−
	2½	−	+	+
	2	−	+	+
	1½	+	+	+
	1	+	+	+
	½	+	+	+
Aluminum pectinate	5	−	−	−
	4	−	−	−
	3	−	+	−
	2½	+	+	+
	2	+	+	+
	1½	+	+	+
	1	+	+	+
	½	+	+	+
Silver pectinate	5	−	−	−
	4	−	−	−
	3	−	−	−
	2½	−	−	−
	2	−	−	−
	1½	−	+	−
	1	−	+	−
	½	+	+	−

TABLE IV

GROWTH OF TEST ORGANISMS IN SOLUTIONS OF URONIC ACIDS WITHOUT ALTERED PH LEVELS

	Ps. aerugenosa					S. aureus					E. coli				
	¼%	½%	1%	1½%	2%	¼%	½%	1%	1½%	2%	¼%	½%	1%	1½%	2%
Glucuronolactone	−	−	−	−	−	−	−	−	−	−	+	−	−	−	−
Galacturonic acid hydrate	+	−	−	−	−	+	−	−	−	−	+	−	−	−	−

TABLE V

GROWTH OF TEST ORGANISMS IN SOLUTIONS OF URONIC ACIDS BUFFERED TO PH 5.5

	Ps. Aerugenosa					S. aureus					E. coli				
	¼%	½%	1%	1½%	2%	¼%	½%	1%	1½%	2%	¼%	½%	1%	1½%	2%
Glucuronolactone	−	−	−	−	−	−	−	−	−	−	+	−	−	−	−
Galacturonic acid hydrate	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+

TABLE VI

DILUTION SERIES OF TEST COMPOUNDS OF SUBLETHEL CONCENTRATIONS IN TRYPTOSE BROTH SOLUTION AND COMBINED WITH STREPTOMYCIN (TEST ORGANISM PSEUDOMONAS AERUGENOSA)

Preparation	Concentration of Streptomycin															
	100μ/cc.	50μ/cc.	25μ/cc.	10μ/cc.	5μ/cc.	2.5μ/cc.	1μ/cc.	0.5μ/cc.	0.25μ/cc.	0.1μ/cc.	0.5μ/cc.	0.025μ/cc.	0.01μ/cc.	0.005μ/cc.	0.0025μ/cc.	Control
A. Control, tryptose broth alone	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
B. 0.5% of 7% methoxyl pectinic acid	−	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
C. 1.5% of 7% methoxyl pectinic acid	−	−	+	+	+	+	+	+	+	+	+	+	+	+	+	+
D. 0.5% of 5% methoxyl pectinic acid	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
E. 1.5% of 5% methoxyl pectinic acid	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
F. 0.5% of bismuth pectinate	−	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
G. 0.5% of aluminum pectinate	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
H. 0.5% of silver pectinate	−	−	−	+	+	+	+	+	+	+	+	+	+	+	+	+
I. 0.5% of galacturonic* acid hydrate	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
J. 0.5% of galacturonic acid hydrate (pH 5.5)	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
K. 0.2% of galacuronolactone (pH 5.5)	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+

* 0.5 per cent of unbuffered galacturonic acid hydrate is bactericidal for this organism. This dilution series was included, however, to illustrate again the difference in the action of buffered and unbuffered galacturonic acid.

content was consistently the most bactericidal. It would also seem that glucuronic acid was slightly more potent in unbuffered solution than was galacturonic acid but, when adjusted to a pH of 5.5 with sodium hydroxide, there was a very noticeable difference between the antibacterial properties of the two compounds. At the adjusted pH level the galacturonic acid permitted heavy growth of the test organisms in all concentrations. This was in agreement with the findings of Steinhaus and Georgi.[9] The glucuronic acid, however, remained as effective in killing the test organisms at pH 5.5 as before neutralization although at the higher pH considerable of the acid was converted into sodium glucuronate.

With the aforementioned facts established it was then necessary to find whether the addition *in vitro* of several antibiotics in varying amounts to sublethel broth concentrations of pectins, pectinates and the uronic acids produced any such remarkable bactericidal effects as when these compounds were given orally with streptomycin. Therefore, eleven types of dilution series were set up with each type set against all possible combinations of the three test organisms and the four antibiotics, namely, streptomycin, penicillin, bacitracin and tyrothricin. Since this amount of tabular material cannot be presented, a single, representative, eleven-series' table is shown in Table VI. In this table the poor effectiveness of streptomycin against the Ps. aerugenosa is due to the low pH of the solution, at which levels streptomycin loses much of its potency.

From Table VI it may be seen that the combination of the streptomycin with test compounds did produce a mild degree of synergism even though the test substances were all well below their bactericidal concentrations. This synergism did not, however, approach that which functions clinically. Approximately the same degree of synergism existed with penicillin, bacitracin and tyrothricin as was found with streptomycin. There was no change in the effective spectrum of any of these antibiotics due to their combination with the test compounds. Organisms resistant to a given antibiotic showed no change in that resistance due to combination with either the pectins, pectinates or uronic acids.

It is interesting that the test organism found to be most affected by these compounds was the Ps. aerugenosa. This organism has always been a difficult one with which to work both clinically and in the laboratory.

COMMENTS

It would appear that the work of Mast and Harrison[1] offers the first opportunity for almost completely sterilizing the contents of the lower colon. Whether or not higher levels of the gastrointestinal tract are similarly sterilized is now being studied. Repeated publications by Lockwood[10,11] as well as others have shown that streptomycin alone could not accomplish such sterilization.

As was said before, there have been much controversial data regarding the antiseptic power of the pectins, pectinates and uronic acids employed in this present work. It is hoped that this study will clarify that portion of the problem and leave the way open for further investigation as to the synergism which operates when these two widely different classes of compounds are combined in the gastrointestinal canal.

Needless to day, a technic by which any or all of the gastrointestinal tract can be sterilized offers numerous possibilities for further study. The physiologic effect on the host, the more satisfactory preparation for gastrointestinal surgery and the further study of conditions in which enteric autointoxication has been held as an etiologic factor are only a few of these. Such studies are now in progress by the authors at this and associated institutions.

CONCLUSIONS

1. Explanatory data are presented regarding the ability of streptomycin given

together with either pectins, pectinates or uronic acids to sterilize the contents of the lower colon.

2. The chemistry and biochemistry of the pectins, pectinates and uronic acids are briefly discussed and data are presented to show that these compounds possess varying degrees of antibacterial potency.

3. It is shown that only a slight degree of synergism exists *in vitro* between these compounds and streptomycin as well as with other antibiotics. No such degree of synergism exists *in vitro* as apparently is the case in the human gastrointestinal tract.

4. Discussion is presented indicating the importance of a technic which will permit sterilization of all or part of the gastrointestinal canal.

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Streamlined Articles

CAROTID BODY TUMORS

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NOT many surgeons have operated upon or are acquainted with the clinical picture of carotid body tumors. Only 293 cases have been reported in the literature up to the present. Because of the rarity of the condition a critical review of the literature was made and an additional case associated with carcinoma of the urinary bladder reported.

The diagnosis of carotid body tumor is seldom made or even entertained. They are symptomless tumors and of many years' duration. Fifteen per cent of these tumors are malignant. The majority of them envelop the carotid arteries and jugular vein. The treatment of choice is surgical removal. The magnitude of the surgical procedure is influenced by the necessity of ligation of one, two or three carotid vessels. This procedure, however, increases the mortality and morbidity of the patient. Radiation therapy is recommended for inoperable cases.

It is hoped that this article will stimulate further thought on the subject of carotid body tumors.

* * * *

In 1929 Bevan and McCarthy[1] stated that very few surgeons have operated upon more than one patient, a few have operated upon two patients and no one has operated on more than three. This statement generally still holds true, with certain exceptions. Since that excellent article was written, Rankin and Wellbrock[2] reported twelve cases in 1931. Hertzler[3] reported seven personal cases in 1937. Harrington et al.[4] reported nineteen cases in 1941 and Lahey and Warren[5] reported eighteen cases in 1947. The aforementioned articles and that of Keen and Funke[6] have formed a very sound foundation for the further study of tumors of the carotid body.

Carotid body tumors retain the shape, lobulation and encapsulation of the normal carotid body as well as the histology, but on a larger scale. They vary in size from a hazelnut to an orange. The largest tumor described by Reid[7] weighed 190 Gm. Firm pressure will diminish its size by forcing the blood out of the highly vascular mass. Some writers described the tumor grossly as resembling a kidney while others compare it to the thyroid gland. The tumor is almost always unilateral although eight bilateral cases were reported. Lund[8] mentions a case in which a second tumor appeared in the opposite side of the neck twenty-nine years after removal of the first one. The right and left sides are equally involved. Male and female are almost equally affected. The oldest patient reported in the literature was seventy-three and the youngest seven; the average age is between thirty and forty years. Heredity does not influence the growth of these tumors although Chase[9] reports two members of the same family with carotid body tumors.

The tumor is of a firm, rubbery consistence. The surface is smooth although in some instances it may be nodular. The color varies from grey-red to purple-red. It is not attached to the skin but is fixed to the underlying structures. It is freely movable from side to side but not from above downward. It practically always

pulsates but the pulsation is not that full expansion and contraction of an aneurysm but a transmitted pulsation from the carotid vessels. In some cases the tumor may bulge into the pharynx while in others it may extend to the base of the skull. As the tumor grows in size, it envelops the carotid vessels and jugular veins in the majority of cases; or it may involve or press upon the glossopharyngeal, hypoglossal, vagus, phrenic or superior cervical sympathetic.

Very few cases of malignancy of carotid body tumors are reported. Bevan and McCarthy state that unless tumor cells can be demonstrated invading the tumor capsule or vessel walls with or without regional lymph node involvement, it is very difficult, if not impossible, to make a diagnosis of malignancy. They state that not more than 15 per cent of the tumors are malignant. Rankin and Wellbrock reported a case of bilateral carotid body tumor that was malignant on the left side and benign on the right. Cragg[10] reported a case of concurrent tumor of the left carotid body and bilateral Zuckerkandl's bodies.

The malignant tumors rarely, if ever, result in distant metastasis. Ewing[11] states, "General metastases are not observed." This opinion is also held by Willis.[12] Local extension with involvement of the regional lymph nodes is not uncommon in malignancy of the carotid body. Recurrence may take place even in benign tumors if they are not completely removed.

The clinical diagnosis of carotid body tumor is seldom made or even considered as a differential diagnosis in tumors of the neck. It is a symptomless tumor which occurs in the upper anterior cervical triangle and which may exist for many years. The average duration of the tumor before the patient came to the surgeon complaining of a swelling in the neck was 5.7 years. The longest time was thirty-five years. Symptoms may be present when the tumor bulges into the pharynx or presses on the vagus, cervical sympathetic or recurrent laryngeal nerves. Pressure symptoms may be manifested by headache, dizziness, hoarseness, cough, dysphagia or fainting spells. Stokes-Adams syndrome and Horner's syndrome have been reported in several cases.

The differential diagnoses in order of importance and frequency are metastatic tumors, lymphoma, tuberculoma, neurofibroma, branchial cysts, aberrant thyroid and aneurysm. Bronchogenic cysts are most likely to resemble clinically tumors of the carotid body. If clinical evaluation fails to establish a diagnosis and aneurysm is definitely ruled out, a biopsy would not only establish a diagnosis but would also dictate the mode of treatment.

The treatment of choice of tumors of the carotid body is complete surgical removal without ligation of the carotid vessels if possible. The magnitude of the surgical procedure is influenced by the necessity of ligation of one, two or three carotid vessels. This in turn depends on the fixation of the vessels to or into the tumor substance. In many instances the tumor completely envelops the vessels and fuses with them making their dissection from the tumor impossible. Important nerves of the neck may be similarly incorporated into the tumor substance although not as frequently. Ligation of the common carotid artery in adults, particularly those who are in the latter half of middle age or beyond, is an extremely serious procedure which carries with it a high rate of mortality, 30 to 65 per cent (Rankin and Wellbrock). In their series of twelve cases it was necessary to ligate the common carotid artery in three cases. All of the patients died within the first forty-eight hours. Harrington et al. report a mortality of 20 per cent which they attribute in most part to hemiplegia. The average age of the patients who died following ligation was fifty-three while the average age of those who survived ligation was thirty-one. In Keen and Funke's twenty-six cases four patients died from pneumonia following resection or division of the vagus nerve and five died from hemiplegia, aphasia and

acute edema of the lungs. In three the voice was affected.

Phelps, Case and Snyder[13] reviewed 154 cases of which 148 patients were operated upon with a mortality of 24 per cent. The mortality was 30 per cent in the group of patients in which carotid ligation was necessary. Seven per cent of those who recovered following ligation of the carotid artery had cerebral complications.

According to Dickenson and Traver in 50 per cent of the reported cases it has been necessary to sacrifice the common carotid artery which has resulted in the death of 30 per cent of the patients. Half of those who survive will have permanent brain damage.

Keen comes to the following conclusion: "The symptoms and inconvenience of the tumor of the carotid body are generally so slight that I do not think any patient who is not suffering seriously or where the tumor does not exhibit symptoms of malignancy or a rapid growth should be subjected to the grave dangers which such operation may involve. Surely no prudent surgeon would be willing to cause his patient to face such dangers simply for cosmetic purposes. If, on the other hand, the tumor is growing rapidly and presents a malignant appearance, then I believe operation is justified, but not otherwise."

Considering the difficulty in diagnosing carotid body tumors, the clinical uncertainty of their malignant nature and the fact that carotid ligation is not always necessary, one would rather agree with the conclusion of Lahey and Warren that "all laterally located, discrete and movable tumors of the neck should be explored." Only through exploration and the taking of a biopsy can one determine not only the nature of a primary tumor of the neck but the operability and prognosis. Bevan and McCarthy believe that neoplasms of the carotid body should not be removed when it is necessary to ligate the common carotid artery in order to complete the operation. They recommend local anesthesia as best for this operation.

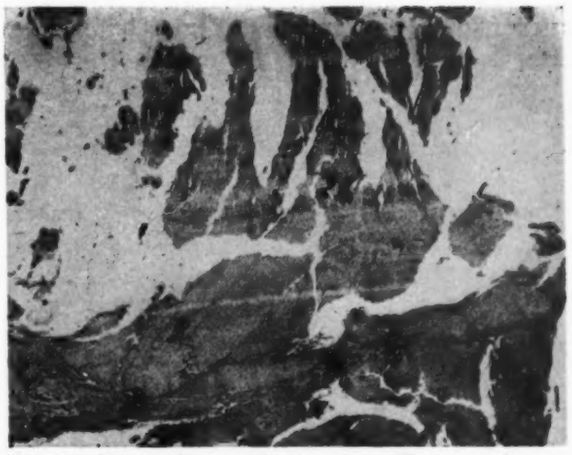

FIG. 1. Photomicrograph shows papillary carcinoma of the urinary bladder. × 50.

Harrington et al. recommend preoperative systemic compression of the common artery against the transverse process of the sixth cervical vertebra several times a day for a few weeks, with the hope of developing collateral circulation in the brain on the affected side. Compression should be carried out for gradually increasing periods until the patient can tolerate complete compression of the vessels for a long period without experiencing faintness or loss of consciousness.

Lahey and Warren state that the common carotid artery should be gradually compressed until the patient can tolerate complete compression for a period of ten minutes three times a day without any symptoms. They also state that if the common, internal and external carotids are ligated, it should be accompanied by ligation of the internal jugular vein.

Radiation therapy in inoperable carotid body tumors has not been given a sufficient trial to form a conclusion as to its application. Bevan and McCarthy report good results in one case of carotid body tumor that was explored but not removed. Lahey and Warren report three cases in which patients were treated with radiation therapy resulting in slight reduction in the size of the tumor in each instance. Phelps, Case and Snyder, on the other hand, report seven proved cases of carotid body tumors treated by radiation without appreciable

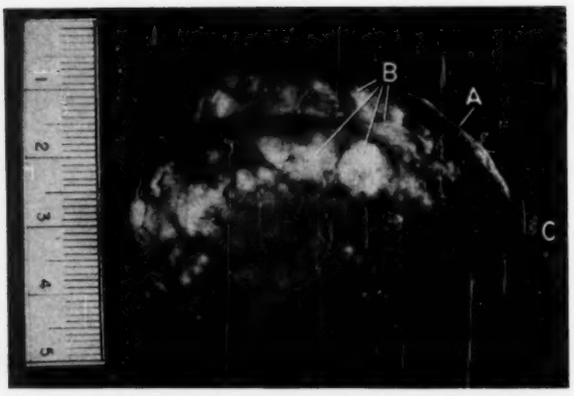

FIG. 2. Gross specimen of the carotid body tumor shows capsule (A) with its septi dividing it into lobules (B) and terminating at the hilus in a pedicle, the ligament of Mayer, (C).

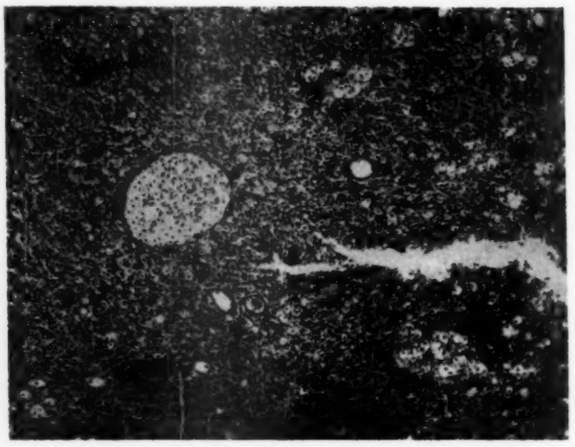

FIG. 3. Photomicrograph shows the histologic structure of the tumor. × 140.

results. One may therefore conclude that if a carotid body tumor is explored and found inoperable, radiation therapy may be given a trial.

CASE REPORT

The patient was first seen on October 11, 1943, in the elevator as he went up to be admitted for carcinoma of the urinary bladder. When questioned about the swelling on the left side of his neck, he replied unconcernedly, "Oh, that! That is nothing. I have had it more than two years and it does not bother me."

The patient was a white male, aged seventy-two, weighing 119 pounds. He was in fairly good condition. His blood pressure was 170/100. He was transferred to the Brooklyn Cancer Institute from another hospital with a diagnosis of carcinoma of the urinary bladder. No mention was made on the transcript about a tumor of the neck. His past history and family history were essentially negative except for a dry, non-productive cough of two years' duration.

The tumor on the left side of his neck was about the size of an orange, extending from the angle of the mandible to the upper border of the clavicle and partially covered by the sternocleidomastoid muscle. It was of a soft, rubbery consistency, somewhat nodular, partially fixed to the underlying structures but not to the skin. It was somewhat movable from side to side but not from above downward. The skin appeared intact. A transmitted pulsation was present but no bruit. Slight traction on the tumor produced a cough similar to the one he had had for the past two years. Roentgen and fluoroscopic examination of the neck only corroborated the diagnosis of a soft tissue tumor. The chest x-ray was negative.

The biopsy of the tumor in the urinary bladder was reported as papillary carcinoma. (Fig. 1.) The cardiogram showed myocardial damage with occasional premature contraction. The red count was 3,380,000 with 72 per cent hemoglobin; the white count was 14,000 with 80 per cent polymorphonuclears, 17 lymphocytes and 3 per cent monocytes. The blood urea 24 mg., creatinin 1.9, sugar 100, cholesterol 170, serum acid phosphatase 3 and serum alkaline phosphatase 6.8; the blood Wassermann test was negative.

On October 20, 1943, the patient was taken to the operating room. Under local anesthesia of 1 per cent novocain an incision was made along the anterior border of the sternocleidomastoid from the angle of the mandible to the upper border of the clavicle. A soft, encapsulated nodular tumor was found extending from the angle of the mandible to 2 cm. beneath the clavicle. The capsule appeared to fuse with the carotid sheath and was surrounded by dilated veins and fine nerve fibers. It was only partially adherent to the carotid vessels. When the carotid sheath was opened, the tumor was found to be attached by a pedicle to the crotch of the bifurcation of the common carotid artery. The tumor with its pedicle simulated a kidney with its ureter emerging from the pelvis. (Fig. 2C.)

The tumor was carefully freed from the surrounding structures by dull and sharp dissection. The tumor pedicle was first loosely clamped and the patient's reaction observed.

It was then doubly clamped, cut and ligated. The tumor was then removed in its entirety. Complete hemostasis was established and the wound was closed. The histologic report confirmed the diagnosis of carotid body tumor. (Fig. 3.)

The patient made an uneventful recovery. Ten days later the urologist fulgurated the bladder carcinoma and inserted radon seeds.

In August, 1944, the patient developed a moderate amount of hoarseness and laryngoscopic examination revealed a partial paralysis of the right vocal cord. The patient was then observed in the Outpatient Department at frequent intervals. In August, 1945, he was admitted to another hospital where he died from uremia. There was no recurrence of the tumor of the neck.

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ULTRAVIOLET BLOOD IRRADIATION THERAPY (KNOTT TECHNIC) IN THROMBOPHLEBITIS*

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SINCE the first report on the use of ultraviolet blood irradiation therapy in thrombophlebitis[1] was published, we have used this method in an additional eighteen cases of thrombophlebitis; of these eighteen, ten were acute and eight chronic. As a result of our experience and observations in these eighteen cases as well as the original thirteen we have come to the following conclusions:

Following the use of ultraviolet blood irradiation in *acute* thrombophlebitis we found that *pain* was the first symptom to disappear, the time of disappearance normally varying from two to twelve hours; very often some slight pain persisted as long as seventy-two hours. *Tenderness* along the course of the affected vein usually disappeared within two to six hours after all pain had subsided. *Fever*, when present, dropped to normal within forty-eight hours. If a concomitant suppurative process was present, the temperature usually remained elevated between 99° and 101°F. until drainage was effected. *Edema* was the last symptom to disappear, usually doing so one to twelve days after treatment was started. When present *coldness* of the affected extremity, insofar as the patient's observation could be relied upon, was relieved within eight to forty-eight hours.

Following the use of ultraviolet blood irradiation in *chronic* thrombophlebitis we found that *pain* was the first symptom to disappear, the time of disappearance ranging from forty-eight hours to two months. *Tenderness* along the course of the affected vein disappeared in time periods ranging from two days to two months following initial ultraviolet blood irradiation. *Fever*, when present, was usually low grade and returned to normal within forty-eight hours after the first treatment. *Edema*, the last symptom to disappear, usually did so five days to two months following initial therapy. One patient with scar tissue present from previous surgery had no marked reduction in edema although all other symptoms disappeared.

In general the longer the patient had had thrombophlebitis before blood irradiation the longer he or she required before the various symptoms disappeared entirely.

* * * *

The blood irradiation treatments given to these patients were made possible by the use of the Knott technic and the Knott hemo-irradiator.[2,3] In this procedure a predetermined amount of blood was withdrawn from an antecubital vein (1.5 cc. per pound of body weight) and passed through the Knott hemo-irradiator. The latter is a precision machine which exposes the withdrawn citrated blood at a predetermined rate to ultraviolet rays from a water-cooled quartz mercury vapor lamp and returns the irradiated blood to the patient through the needle use for the initial venipuncture.

CASE REPORTS

CASE 1. A twenty-five year old white female developed thrombophlebitis of both femoral veins following an uneventful delivery of a normal child. Her temperature was 103°F., pulse 160, respirations 40, white blood cells 30,000 and polymorphonuclears 92 per cent. With her phlebitis she complained of marked pain and tenderness in both legs and bilateral edema. Forty-eight hours following one ultra-

* Aided by a grant from the Madison Foundation for Biochemical Research, New York, N. Y.

violet blood irradiation her temperature was 98.8°F., pulse 120, respirations 24, white blood cells 21,000 and polymorphonuclears 82 per cent. Pain, tenderness and edema had completely disappeared and subsequent convalescence was uneventful.

CASE II. A thirty-five year old white male was first seen September 9, 1947, as an outpatient. He gave a history of chronic thrombophlebitis in both lower extremities with deep involvement of both saphenous and femoral veins of twelve years' duration. His temperature was normal; he had pain and tenderness along the route of both saphenous veins and marked bilateral edema. Prior to seeing us he had run the gamut of treatment from bed rest, sulfa drugs and penicillin to surgery, including scarification and bilateral vein-stripping. After his initial ultraviolet blood irradiation all tenderness disappeared in thirty-six hours. Pain was noticeably less in twenty-four hours and was completely gone in four weeks following the initial ultraviolet blood irradiation. Swelling has not markedly disappeared mainly, we believe, as a result of scar tissue following previous surgery but it has receded greatly, appearing only while the patient is standing or walking. Since this treatment he has had one exacerbation of pain, fever and tenderness. This, however, was easily controlled within two days with the use of ultraviolet blood irradiation. Since that time, March, 1948, he has had no exacerbation, has returned to full activity and is complaint-free.

SUMMARY

1. In eighteen consecutive instances patients with acute or chronic thrombophlebitis were given ultraviolet blood irradiation therapy (Knott technic).

2. In the ten acute cases there was a rapid subsidence of pain and tenderness within twelve to forty-eight hours. Fever, when present, returned to normal within forty-eight hours and the edema usually disappeared by the twelfth day.

3. In the eight chronic cases the symptoms also disappeared but over a generally longer period of time, depending on the duration of the disease process prior to treatment.

4. There were no noticeable untoward effects either immediate or delayed.

5. The original findings are confirmed and extended.

6. The Knott technic of ultraviolet blood irradiation has been found a safe and efficient method of controlling acute and chronic thrombophlebitis.

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SIX PRIMARY CARCINOMAS IN ONE PATIENT*

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A CASE is reported in which there were six separate primary carcinomas involving respectively the jejunum, the transverse colon, the sigmoid colon, the rectosigmoid colon, the urinary bladder and the skin.

* * * *

CASE REPORT

A fifty-three year old Caucasian male was hospitalized on February 11, 1947, because of severe stabbing pain in the right lower quadrant and perineum of six hours' duration.

He had been in good health until 1943, at which time he had developed hematuria; a bladder tumor had been excised through a suprapubic cystotomy. Thereafter the patient had no further complaints until the beginning of January, 1947, when he sought admission to another hospital where a cystoscopy was performed and a bladder tumor removed by fulguration. Both specimens were diagnosed as transitional cell carcinoma grade II.

Physical examination disclosed a well nourished, apprehensive male who appeared to be acutely ill and in pain. The abdomen was distended and tender in the right lower quadrant, with rebound tenderness throughout. Peristaltic activity was within normal limits; no masses were palpable. Rectal examination was negative. The patient's temperature was 101.6°F. by mouth. There was a leukocytosis of 16,300 with 89 per cent polymorphonuclear cells. The hemoglobin level was only 11.7 Gm. and the red cell count was 3.6 million. Urinalysis revealed thirty to forty pus cells per high power field. The first stool was soft and brown and manifested a strongly positive test for occult blood. X-ray of the abdomen revealed a moderate amount of gas in the small and large intestines.

With bed rest and parenteral feedings the pain and fever subsided over the course of three days. However, on the fifth day the patient had another seizure of colicky pain in the right lower abdomen associated with a temperature rise to 103.4°F. Roentgen examination of the abdomen showed gaseous distention of the small intestine with intralumenal and extralumenal fluid. There was no gas visible in the large intestine. A diagnosis was made of intestinal obstruction due probably to an acute inflammatory process in the region of the terminal ileum.

At operation the appendix was found to have perforated into the pelvis; diffuse peritonitis involved the lower half of the peritoneal cavity. An appendectomy was performed. During the course of the operation a rosette-shaped mass 2½ cm. in diameter was palpated in the small intestine. A tentative diagnosis of carcinoma was made but excision was deferred in the presence of spreading peritonitis. The operative convalescence was complicated by a pelvic abscess which was drained through the lower abdominal wall.

During the postoperative recovery further studies were undertaken to determine the nature of the lesion in the small intestine. A gastrointestinal series with small bowel studies failed to demonstrate any abnormality. However, a barium enema disclosed two deformities of the large intestine, one in the right half of the transverse colon and the other in the proximal portion of the sigmoid colon. Both deformities had an appearance suggestive of carcinoma but in the presence also of a small intestinal lesion they were thought to be inflammatory in nature. The colon was normal up to 20 cm. from the anus by sigmoidoscopic examination. Stool studies for intestinal parasites were negative but the stools contained occult blood on a meat-free diet. During the convalescence the patient had daily afternoon rises of temperature to 99.4°F. and an elevated

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sedimentation rate. There was a persistent hypochromic microcytic anemia which did not respond to iron therapy. Occasionally the patient complained of left lower quadrant abdominal discomfort relieved after an enema or the passing of flatus. The patient was on a full diet but there was no consistent weight gain. Although amebiasis had not been demonstrated in repeated stool examinations, the clinical picture was considered suggestive of amebic granuloma. The patient received two courses of emetine, each lasting seven days, and one course each of diodoquine and carbarsone. There was no apparent improvement.

On June 11th, four months after admission, celiotomy was performed. In the jejunum 15 cm. from the ligament of Treitz a hard, constrictive lesion measuring 3 cm. in its greatest diameter was found. The jejunal mesentery contained a group of enlarged lymph nodes, the largest measuring 1½ cm. in diameter. Their consistency and appearance were characteristic of metastatic carcinoma. In the right side of the transverse colon a short distance from the hepatic flexure there was an annular constrictive mass of stony hard consistency, 5 cm. in length and 3 cm. in width, covered by intact serosa. There was no associated lymphadenopathy. The proximal portion of the sigmoid colon contained an annular constrictive tumor measuring 6 by 5 cm. in area invading the serosa. There was mild lymph node enlargement in the sigmoid mesentery. Twice during the operation before resection was attempted the patient manifested a sharp drop in blood pressure and a rapid, thready pulse. Because of the patient's precarious condition the operation was restricted to a resection of the jejunal lesion and attached mesentery and an exteriorization of the neoplasm of the transverse colon by a modified Paul-Mickulicz procedure. Five days later the exteriorized mass was excised by cautery.

On July 17, 1947, the cancer of the sigmoid colon was resected and an end-to-end anastomosis was performed. At this time the remainder of the pelvic colon was normal to palpation.

The patient returned in January, 1948, for closure of the transverse colostomy. The two colostomy openings were dissected from the abdominal wall and the peritoneal cavity explored prior to reanastomosis. There was no evidence of metastasis in the liver or at any of the sites of previous anastomosis. Several suspicious areas surrounding the jejunal anastomosis were biopsied and found free of cancer. However, a hard mass was found in the rectosigmoid colon about 6 cm. above the peritoneal reflection of the rectum; the serosa was intact and the mesenteric lymph nodes were not enlarged. Inasmuch as a permanent colostomy was probable, the previously exteriorized portion of the transverse colon was removed and a double loop colostomy made. The abdomen was then re-entered through a transverse incision in the left lower quadrant and the rectosigmoid colon excised. The proximal and distal segments of the intestine were closed and peritonealized.

In July, 1948, the patient had a sudden onset of left lower quadrant pain accompanied by nausea and vomiting. At operation a perforated ulcer on the anterior surface of the jejunum 2 cm. distal to the gastrojejunostomy was repaired. Later a small nodule developed at the site of pressure from a colostomy belt. This was thought to be a furuncle and was treated with warm moist compresses. Because the nodule grew larger and firmer under this regimen, it was excised for histologic study and was found to be a squamous cell carcinoma.

When seen two years after the jejunal resection, the patient manifested no clinical or laboratory evidence of persistence or recurrence of any of his carcinomas.

Microscopic examination of the tumors showed each to be a primary carcinoma with a distinct histologic structure. The jejunal tumor (Fig. 1) consisted of papillary masses of highly anaplastic, rapidly growing cuboidal to columnar cells invading the mucosa and submucosa. There were large areas of necrosis with infiltration by polymorphonuclear leukocytes. The mesenteric lymph nodes presented a similar microscopic appearance. The diagnosis was papillary adenocarcinoma of the jejunum with metastasis to the regional lymph nodes.

The excised portion of the transverse colon (Fig. 2) contained a tumor composed of columnar cells forming well differentiated glands invading the submucosa and muscularis. Mitotic figures were relatively few. A diagnosis of ulcerated adenocarcinoma of the transverse colon was made.

Sections of the sigmoid colon (Fig. 3) showed a malignant lesion composed of irregular glandular acini and alveoli of tumor cells invading the entire wall of the bowel. Although

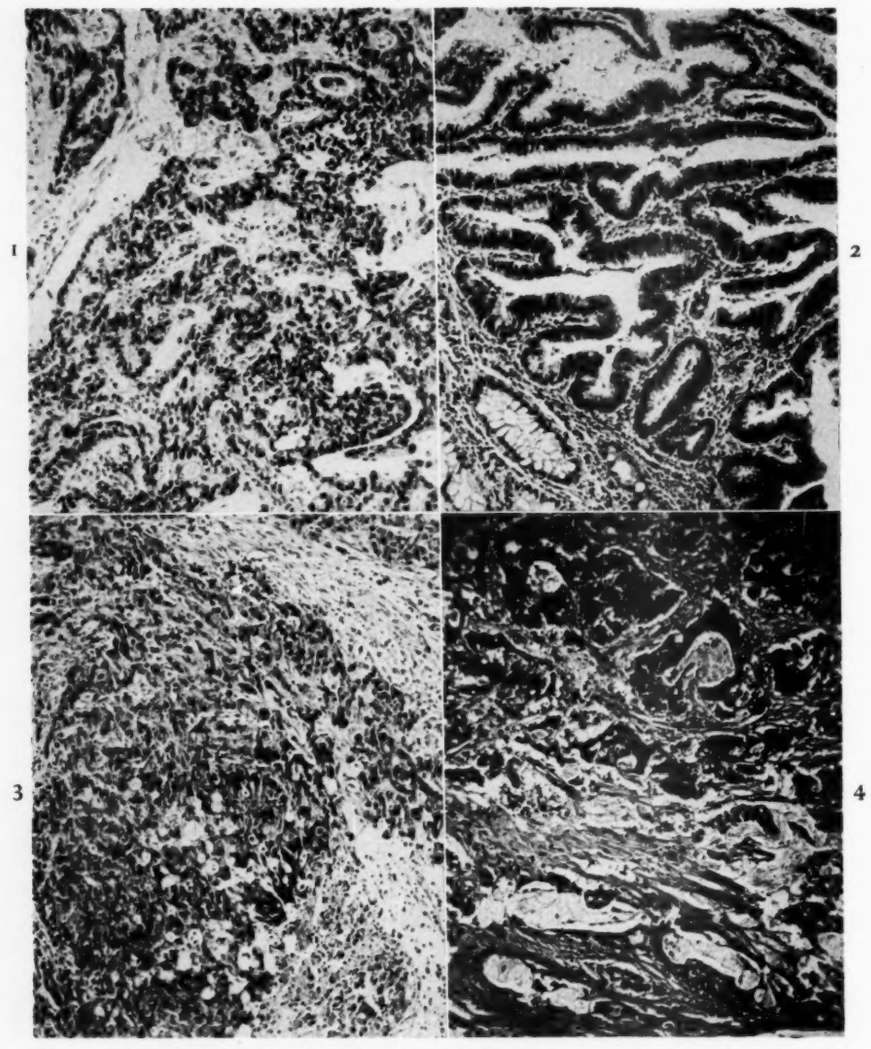

FIG. 1. Anaplastic adenocarcinoma of jejunum consisting largely of solid masses of poorly differentiated cells with some tendency to form acini; some mucus production.
FIG. 2. Well differentiated adenocarcinoma within the submucosa of the transverse colon.
FIG. 3. Undifferentiated carcinoma of the sigmoid colon showing plaques and small acini with many signet-ring tumor cells containing mucus.
FIG. 4. Anaplastic adenocarcinoma of the rectosigmoid within the muscularis; note abundant mucus.

fairly cellular masses of signet ring tumor cells were found in places, some portions of the tumor consisted almost entirely of mucoid material. Mucus-producing adenocarcinoma of the sigmoid colon was the diagnosis.

The lesion of the rectosigmoid colon was formed by irregular glandular structures which invaded the muscularis and extended into the serosa. The lining cells were highly anaplastic and contained abundant cytoplasm and much mucoid material. There was also a large amount of mucus between the bundles of muscle fibers. Although both carcinomas of the sigmoid colon contained a great deal of mucus, their microscopic pattern was distinctly different. (Figures 3 and 4.)

The tumor removed from the skin was described as a well differentiated keratinizing squamous cell carcinoma.

COMMENTS

This patient entered the hospital because of symptoms due essentially to acute appendicitis; otherwise, he might not have sought medical assistance. Cecal distention

secondary to partial obstruction of the distal colon[1] may well have entered into the etiology of the appendicitis. However, the jejunal cancer, although more extensive than the others, was apparently asymptomatic and was detected only by chance in the course of an unrelated operative procedure. Even after the presence of the small bowel lesion was known, roentgen studies failed to reveal it.

The two colonic lesions which were detected by barium enema examination confused the picture in the known presence of a small intestinal lesion. Despite an X-ray appearance which was rather characteristic of carcinoma, the simultaneous occurrence of these cancers in the small intestine, transverse colon and sigmoid colon seemed so unlikely that every possible device (including a therapeutic test with anti-amebic drugs) was used to prove that the lesions were granulomas.

All four intestinal carcinomas were separate primary growths, each with a distinctive histologic picture and all quite different from the transitional cell cancers previously removed from the bladder. Warren[2] has pointed out that the statistical expectancy of multiple primary malignancy should be only 5 or 6 per 1,000 whereas the observed incidence is 37 per 1,000. From this he concludes that there is a significant element of susceptibility involved in the etiology of malignant neoplasms. In this case it seemed that the patient's tissues were especially prone to undergo malignant degeneration. The last tumor removed had encircled the bowel and extended into the serosa yet had not been palpable six months previously.

Frequently, as in this instance, the simultaneous occurrence in one patient of two or more unrelated neoplasms obscures the diagnosis because of the impression that primary malignancy is usually single. Actually the occurrence of one or more additional primary neoplasms in a patient already afflicted with one such lesion is six or seven times as likely as the first occurrence of a malignancy in an unafflicted individual. Multiplicity should be regarded as confirmatory rather than contradictory evidence of malignancy.

Acknowledgment. The authors wish to express their gratitude to Dr. Perry J. Melnick and Dr. Irving Reingold for their interpretation of the gross and microscopic pathology.

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CARCINOMA OF CECUM IN YOUNG MAN OF TWENTY-ONE

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IN large bowel surgery a few words are apropos. No colon surgery should be done until the patient who is often debilitated and whose nutrition may be poor has been brought as close to normal status as possible. This can be accomplished by the administration of amino acids, blood transfusions and vitamin therapy. The use of sulfasuxidine or sulfathalidine which reduces the bacterial count of the bowel and renders the organisms less virulent (thus lowering the mortality of colon surgery) is an essential requirement preoperatively. In this case it would have been unwise to do any colon surgery at the time of the original operation because the bowel was not prepared properly with sulfasuxidine or irrigations.

* * * *

CASE REPORT

A young man of twenty-one came into the hospital doubled up with pain. He appeared acutely ill and was groaning. He gave a history of intermittent pain for four months in the right lower quadrant which had become severe in the past four hours. There was no change in bowel habits.

Examination revealed tenderness and marked spasm in right lower quadrant. No mass could be palpated because of the spasm present. The laboratory report was as follows: white blood cells, 19,000; 91 per cent polymorphonuclears on admission; red blood cells 3,800,000; hemoglobin 80 per cent.

The condition of the patient was diagnosed as an acute exacerbation of a chronic appendix and operation decided upon. A right lower paramedian incision was made and the abdomen opened. On opening the abdomen, instead of an acute appendix, a large, firm mass involving the cecum was found. It looked and felt like a malignancy and there was some swelling and edema of the mass. It was thought advisable not to do anything at this time because of the nature of the lesion and because the bowel was not prepared for a formidable procedure. The abdomen was closed without drainage.

The patient was then prepared for two weeks with intravenous amino acids, intravenous fluids and sulfasuxidine, and multiple bowel irrigations before surgery.

With the patient under spinal anesthesia and with an intravenous drip going, a right paramedian incision was made from the costal margin downward toward the inguinal ligament. On opening the abdomen it was found that several adhesions had formed between the mass and the abdominal wall since the previous celiotomy. The liver showed no metastases. The mass was quite large and there were fairly large glands in the mesentery of the cecum but pathological examination showed these to be inflammatory. Then, because the patient was in good condition and because there was no obstruction in the tumor, a one-stage ileocolic resection using the Rankin clamp was performed with an end-to-side anastamosis between the ileum and the transverse colon.

During and immediately after operation the patient received a total of 1,000 cc. of citrated blood. Postoperatively, a Wangensteen suction was kept down for about three days prophylactically to keep tension off the anastomosis. The patient received about 3,000 cc. of intravenous fluids daily including amino acids and vitamins parenterally. After that he was started on water, then liquids and later soft diet. The wound healed *per primam* and the patient was out of the hospital in ten days.

Since operation the patient has gained 25 pounds in weight and has had no complaints.

The pathological diagnosis was adenocarcinoma of the cecum.

SUMMARY

The preceding case was presented because the subject was relatively young for carcinoma of the colon and because of the method employed in handling the patient.

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Case Reports

DESMOID TUMORS OF THE ABDOMINAL WALL

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DESMOID tumors are of interest because they are uncommon and frequently are a problem in differential diagnosis. These tumors are important because they can be excised surgically with permanent cure if all of the tumor is removed. Miller (1838) first used the word "desmoid" to describe these benign tumors which arise from musculoaponeurotic structures. The word is well chosen as it represents a contraction of two Greek words, "desmos," a band or tendon, and "eidos," appearance.

Desmoids may develop in any of the striated muscles or their aponeuroses, for instance, in the pectoralis major, sternocleidomastoid, digastricus, masseter muscles, scapular region, biceps, extensor carpi ulnaris, rectus femoris, glutei or hamstring muscles. They do not appear in these regions, however, as frequently as they are found in the abdominal wall. This paper deals only with desmoids of the abdominal wall. In this region they occur most frequently in relation to the rectus muscle below the level of the umbilicus. Stone[8] found the right lower part of the abdominal wall involved more frequently than any other portion of the abdomen.

Various authors[2,5–7] give different occurrence rates. Approximately 90 per cent of the reported cases were in women and about 90 per cent of these women had borne children. Although the exact etiologic factor is unknown, Waugh[9] summarizes several observations which seem to indicate that these fibromas result from trauma with hemorrhage; and that the characteristics of a tumor are assumed during the process of organization of the hematoma. The following observations bear this out: (1) frequent history of trauma to the region; (2) the tumors have occurred in the scars of previous incisions and (3) they are most common among women who have been pregnant.

Pathology. The desmoids are densely hard and cut with a grating sound. The cut surface reveals interlacing bands of fibrous tissue. The larger tumors tend to be soft in the center and some are actually cystic. Although Straub[10] reports changes in the overlying skin and keloid formation, this is distinctly unusual.

Microscopically, the appearance is that of a cellular fibroma occurring in striated muscle. Ewing[3] states that not infrequently the structure resembles a neurofibroma and varies from a hard, acellular fibroma to a rather cellular fibrosarcoma. Generally speaking, desmoids are not malignant and, while prone to local recurrence, do not metastasize. Sarcomas should not be included in this group.

The center of the tumor is older than the peripheral portion. At the periphery the tumor frequently infiltrates the surrounding muscle. Waugh and others state that there is no capsule or definite line of cleavage between the tumor and the adjacent muscle. When the tumor is adjacent to a fascial plane, an appearance of encapsulation may result.

The inclusion of striated muscle fibers is an unusual feature of the tumor. From the practical surgical standpoint it is undoubtedly easy to leave infiltrating portions

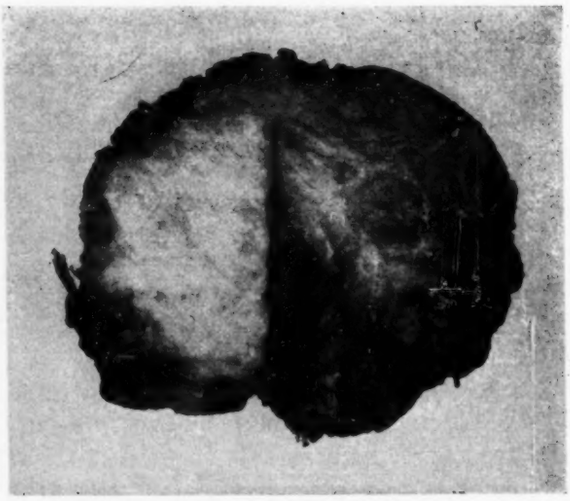

FIG. 1. Gross appearance of desmoid tumor of right rectus muscle.

of the tumor in the surrounding musculature. Pearman and Mayo's[5] recent study has shown that these tumors are definitely benign and that "recurrences" are actually the result of incomplete removal. They found that patients with these "recurrences," when subsequently undergoing complete excision, were cured for years.

History and Examination. Usually the patient finds a small, hard lump in the lower abdominal wall or she may have been totally unaware of the mass which is discovered during the course of a routine physical examination. When the mass gets larger, it may be visible (Smith[4]) or may produce a dragging sensation and occasionally actual pain due to the weight of the tumor. Walters and Church[11] found that only one-fourth of the patients in their series complained of pain or soreness.

Examination reveals a mass fixed in the abdominal wall which does not move with respiration. The mass can be moved to a slight degree with the abdominal muscles relaxed. With these muscles tensed, however, the tumor is completely immovable. The tumor may seem more superficial than the usual intraperitoneal mass. It is not attached to the skin, thus distinguishing it from a keloid.

Treatment. The treatment is wide surgical excision. Radiotherapy alone has not been successful in curing these tumors but may be used as a supplement to surgery. This is particularly desirable when complete removal of the tumor is impossible. Removal of large tumors may leave a defect in the abdominal wall that is difficult to close and which may require fascial transplants

CASE REPORTS

The following patients on whom we operated present some interesting features.

CASE 1. The patient was a thirty-six year old white female who complained of pain in the right side of her abdomen of approximately two weeks' duration. The past history revealed that she was married for eleven years and had two children. One child was nine years old and the second child was two. The remainder of the past history was irrelevant.

About two weeks before seeking medical advice the patient noticed the gradual onset of an aching type of pain in the right side of the abdomen. The pain was continuous and did not radiate. It was more severe in the right upper quadrant than in the right lower quadrant. There was no history of indigestion or selective dyspepsia and the pain was unaffected by food intake.

Examination revealed a short, stocky woman weighing 220 pounds. The abdomen was very obese. A globular, firm, tender mass 6 to 8 cm. in diameter was palpable in the right upper quadrant. Although there was moderate splinting of the abdomen on the right side, the liver edge could be felt about two fingers below the costal margin. Tenderness made deep palpation of the mass difficult. The mass descended slightly with respiration. The blood count was as follows: red blood cells, 4,900,000; hemoglobin, 13 Gm., 82 per cent; white blood cells, 12,800; neutrophiles, 70; segmented polymorphonuclears, 65; stab cells, 5; lymphocytes, 22; monocytes, 6; basophiles 2; shift to the left.

Although the mass was more medial and firmer than usual, before x-rays were taken a tentative diagnosis of hydrops or carcinoma of the gallbladder was made. The patient was hospitalized. Temperature on admission was 99°F., pulse 80. Cholecystography revealed a normally functioning gallbladder without stones. Urine examination showed a trace of albumin.

FIG. 2. High power magnification showing transformation from normal aponeurosis on the right to interlacing bundles of desmoid tumor on the left.

In view of the negative x-ray findings, exploratory operation was decided upon in order to determine the exact nature of the mass. At operation after going through a panniculus adiposis of about 10 cm. in depth, the tumor was finally encountered. It had completely replaced the rectus muscle for about 6 to 8 cm. A wide excision was done and it was necessary to remove portions of the anterior and posterior rectus sheaths as well as the muscle. The peritoneum was opened and the liver was seen to descend below the costal margin. The gallbladder and remaining viscera were normal. The appendix was removed and the defect in the abdominal wall was closed longitudinally in layers with some difficulty using interrupted silk sutures.

The pathologist reported that the growth removed was a desmoid tumor of the abdominal wall measuring 8 by 5 by 5 cm. Figure 1 shows the gross appearance of the tumor. Figure 2 shows a photomicrograph of the edge of the desmoid tumor showing the transition from normal fascia into interlacing bundles of desmoid tumor.

Convalescence was uneventful. The patient was out of bed the day after surgery and went home on the sixth postoperative day.

CASE II. M. M. a twenty-seven year old white female was admitted to the Alexander Blain Hospital, Detroit, Mich. She had noticed a lump in her lower abdomen on the right side six months before admission. Her past history revealed that she had been married five years before and had had one delivery. One year previously she had a uterine suspension done elsewhere through a midline suprapubic incision. The tumor had grown gradually larger during the past few months.

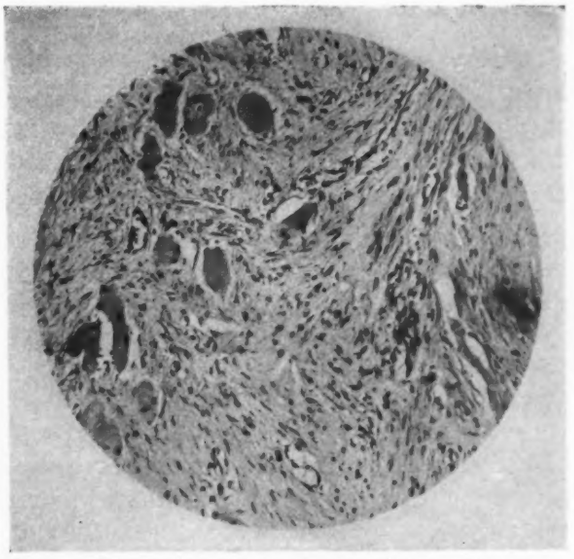

FIG. 3. Photomicrograph of edge of desmoid tumor showing invasion of rectus muscle.

Examination revealed the uterus enlarged to the size of a seven-month pregnancy. There was an irregular, nodular, painless mass measuring 2 by 7 cm. present above the inguinal ligament just to the right of the midline. The skin was movable over the mass. The blood count and urine examinations were normal.

The patient was operated upon with a preoperative diagnosis of tumor of the abdominal wall, possibly malignant. A 6 inch incision was made and the tumor was widely excised including generous portions of the involved fascia. A classical cesarian section was then done. Due to the large defect the abdominal wall was closed with some difficulty. The pathologist described a desmoid tumor measuring 10 by 4 by 4 cm. (Fig. 3.)

The patient's postoperative course was uneventful.

Eighteen months and eight months, respectively, since the dates of surgery both wounds were well healed without any sign of recurrence or hernia.

COMMENT

Whereas most cases of desmoid tumors are painless, it is interesting to note that the first patient had pain and tenderness. The physical signs simulated a hydrops or carcinoma of the gallbladder or an inflamed gallbladder with omentum wrapped around it. The difficulty of differentiating an intraperitoneal mass from a mass in the depths of the abdominal wall by physical examination was insurmountable due to the patient's obesity. At operation the mass was seen to be about 10 cm. below the level of the skin whereas the gallbladder was in direct contact with the deep surface of the tumor.

The second case is of interest for two reasons. A malignant-appearing tumor proved to be benign and was cured by complete radical excision. Cesarian section could be done at the same time.

SUMMARY

Desmoid tumors are frequently preceded by some form of trauma. These tumors are benign and will not recur if excised widely. A case is reported which simulated hydrops of the gallbladder and another which simulated malignant tumor of the abdominal wall.

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CONGENITAL ATRESIA OF THE COMMON BILE DUCT*

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CONGENITAL atresia of the extrahepatic bile ducts has held the particular interest of surgeons since Holmes in 1916 estimated that 16 per cent of the cases reported up to that time were theoretically amenable to surgical therapy. He suggested the establishment of an anastomosis or, if this could not be done, the establishment of an external biliary fistula and later the construction of continuity with the intestinal tract. However, it was not until 1935 when Ladd[1] reported a case in which the patient was successfully treated that this condition was satisfactorily handled. Since that time there have been additional reports of successful surgery[2–11] but not as many as one would expect. In October, 1948, Gray, DuShane and Henegar[2] reported two cases from the Mayo Clinic and could find only fifteen additional cases reported in the English literature. These fifteen included nine individual case reports and six cases reported previously by Ladd and Gross.[3] Also in October, 1948, Leyva and Madigan[11] reported five successful cases from the Children's Hospital of Washington. One of these had apparently been previously reported by Stiegler in 1946[7] and was included in the fifteen mentioned by Gray and his associates. Additional search through the literature has revealed no other cases in which the patient survived for any length of time and these twenty-one cases remain as the only completely successful ones reported.

Chesterman[12] reported a case of atresia of the common duct treated by cholecystoduodenostomy in which the patient died on the ninth postoperative day following a wound separation. Autopsy showed an intact anastomosis. Also, there have been reported cases in which an obstruction due to inspissated bile was relieved by surgery,[9,13,14] the thick bile apparently being on the basis of Rh incompatibility.

Analysis of the twenty-one reported cases reveals that the gallbladder was anastomosed to either the stomach or small bowel in the majority of cases. The procedures used are as follows: cholecystogastrostomy eight, cholecystoduodenostomy six, cholecystojejunostomy one, choledochoduodenostomy three and hepaticoduodenostomy three.

CASE REPORT

A. E., a Chinese-American boy, aged two weeks, was one of twins born in the Reading Hospital on June 18, 1948. It was the mother's first pregnancy and both children seemed normal at birth following an uncomplicated labor. The baby's birth weight was 5 pounds, 4½ ounces. It was noted that the child had the usual amount of "physiologic" jaundice which seemed to lessen about the eighth or ninth day. On the tenth day he was discharged from the hospital but was readmitted the following day because of the development of extreme jaundice and dehydration. The baby's temperature was normal. The abdomen was distended and the liver edge palpable two and a half fingers below the costal margin. There had been no vomiting. The stools were yellowish and the urine dark. The baby's weight was 4 pounds, 15 ounces. Blood count showed hemoglobin 79 per cent, red blood cells 3,800,000 and white blood cells 10,500.

On the twelfth day the weight was 4 pounds, 9 ounces; icterus index was 150 and the serum bilirubin 6 mg. The van den Bergh test showed a delayed direct reaction; the child was Rh positive. He was maintained on 2½ per cent glucose in normal saline, vitamin K and skimmed milk formula, and was admitted to Temple University Hospital on July 2nd, the fourteenth day of life.

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On admission to this hospital the child was extremely jaundiced and dehydrated. There was moderate apathy and skin turgor was poor. The only other positive finding on physical examination was that the liver edge was palpable 4 cm. below the costal margin. The spleen was not palpable and the child's temperature was 98.4°F. per rectum.

A stool on the day of admission was noted as being large, mucoid and pale yellow. Laboratory studies were as follows: hemoglobin 8.7 Gm., red blood cells, 2,710,000, white blood cells 9,200, polymorphonuclears 73 per cent, lymphocytes 20 per cent and monocytes 7 per cent. Coagulation time was four minutes and bleeding time thirty minutes. Urinalysis showed yellow color, no sediment, pH 6.0, negative for albumin, sugar, bile and occult blood. Microscopic study of the urinary sediment showed only 2 to 3 white blood cells per high power field. The blood Wassermann test was negative and the icterus index was 171 units. The van den Bergh direct reaction was very strongly positive and the indirect reaction showed 15.3 mg. per 100 cc. Blood cultures were repeatedly negative.

The child was maintained on a high protein formula with vitamin supplements and was given 10 mg. of vitamin K parenterally daily. On July 3, 1948, he was given 50 cc. of blood; bleeding time was one minute on this date. On July 9th examination of the urine showed a slightly positive reaction for bile pigments and a negative reaction for urobilinogen. The feces were negative for urobilinogen. Blood count showed hemoglobin 11.9 Gm., red blood cells 3,179,000 and white blood cells 18,300, with polymorphonuclears 40 per cent, lymphocytes 45 per cent, monocytes 8 per cent and eosinophiles 6 per cent.

A tentative diagnosis of congenital atresia of the bile ducts was made and the child was prepared for exploration. During this time penicillin was given (10,000 units every three hours from July 2nd to July 8th and 40,000 units every twelve hours from July 9th to July 21st, eight days postoperatively).

Abdominal exploration was done under local anesthesia on July 13th. The liver was somewhat congested. A normal gallbladder was observed; it was distended with dark, sticky bile. The common duct was present and large, and obviously extended down past the insertion of the cystic duct. The common duct was not explored due to the poor general condition of the child. Cholecystojejunostomy was done and the abdomen closed as quickly as possible. During and immediately following the operation 100 cc. of blood were given intravenously.

Postoperatively the child made a satisfactory recovery. Gastric suction was maintained for twenty-four hours and then oral and gavage feedings were begun.

On July 15th, the second postoperative day, the child's temperature was 99.4°F. per rectum and it was noted that a stool showed mixed brownish and yellow color. A blood count showed hemoglobin 13.2 Gm., red blood cells 4,169,000 and white blood cells 9,200; the differential was normal. On July 16th the urine was negative for urobilinogens and on the seventeenth the icterus index had fallen to 86.

During this period the child had a tendency to lose weight but subcutaneous amigens seemed to enable him to begin gaining. On July 23rd the feces were found to be moderately positive for urobilinogen and on this day the skin sutures were removed.

By July 26th the icterus index had fallen to 20.5 units, the van den Bergh direct was slightly positive and the indirect 0.7 mg. per 100 cc. By the thirtieth the wound was well healed.

On August 2nd hemoglobin was 11.5 Gm., red blood cells 4,480,000 and white blood cells 18,400, with a normal differential. By the fifth the icterus index was 19 units, the van den Bergh direct was still moderately positive and the indirect was 0.6 mg. per 100 cc.

On August 12th the child was discharged from the hospital in good condition. Since that time he has been followed up in our Pediatric Clinic. On his last visit to the clinic in October, 1948, four months after operation, he was entirely normal.

COMMENT

Our purpose in presenting this case report is to emphasize the fact that although most children with this condition have proven to be inoperable on exploration, there is a definite salvage; and such patients must be carefully explored before we label them with a hopeless prognosis. When such patients appear to be operable, the simplest possible method should be employed due to the associated debility of the patient.

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BILATERAL SIMULTANEOUS TUBAL PREGNANCY*

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PREVIOUS to 1883 when Lawson Tait[1] of Birmingham, England, operated successfully for a ruptured tubal pregnancy, extrauterine gestation was of interest only from a pathologic view; but through his forceful writings a new impetus was given to its diagnosis and treatment. It should be remembered, as Mr. Tait points out, that the first authoritative treatise on extrauterine pregnancy was written by John S. Parry, an American, in 1876; and antidating Parry, John Bard[2] of New Jersey published a report of three laparatomies for extrauterine pregnancy in 1759. This was followed by the report of William Baynham[3] of Virginia on his two successful operations in 1790 and 1799.

Since Tait published his observations, the literature on extrauterine pregnancy has become so voluminous that a complete review would prove practically impossible.

The occurrence of bilateral simultaneous tubal pregnancy, however, is so infrequent that many texts either omit its discussion or give a short reference to the condition. We believe that an additional report is worthy of record.

The following classification of extrauterine gestation admits of proper grouping: (1) intrauterine pregnancy combined with extrauterine pregnancy; (2) intraperitoneal (abdominal) gestation; (3) multiple pregnancy in a single fallopian tube and (4) coincident pregnancy in each fallopian tube.

The reports of bilateral simultaneous tubal pregnancy have been conspicuous not only because of rarity but also because of the incompleteness of data in some few published reports, so much so, that one is confronted with difficulty in arriving at a correct diagnosis and definite conclusion.[4]

Fishback[2] has published an illuminating résumé of the literature on bilateral tubal pregnancy with emphasis on classification accompanied by a report on multiple tubal pregnancies in which he also found insufficient data in many to be acceptable for definite classification.

Schockaert[6] in 1928 collected from the literature only eighteen cases of simultaneous bilateral tubal pregnancy. The number reported by different observers has varied and many different opinions are expressed regarding the criteria to be used in their classification.

Many explanations have been advanced as to etiology, some plausible others fantastic. Kelly and Noble's[7] summation as to favorable factors seem all inclusive: (1) conditions which interfere mechanically with the downward passage of the ovum and (2) physical and developmental conditions which favor decidual formation in the tubes.

In a search of the literature only eighty-seven reported bilateral simultaneous tubal pregnancies were found to admit of a positive grouping and in only thirty-four could one be positive as to simultaneous conception.

Proust[8] is of the opinion that in practice the diagnosis of bilateral tubal pregnancy is never made preoperatively and most surgeons, I am confident, are in perfect agreement.

CASE REPORT

D. A. Y., a white, married female, aged thirty-four, was hospitalized on September 5, 1947, complaining of abdominal pain and distention. She was the mother of a healthy child of fifteen. A history of five spontaneous

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1A 1B

FIG. 1. Bilateral tubal pregnancy; A, left tube; B, right tube.

miscarriages in the second month during the past fourteen years was obtained. Menstruation began at the age of twelve and was of four or five days' duration, with slight pain; it has been of the thirty-day type. Her last menses occurred in the early days of July, 1947, accompanied with no unusual symptoms.

On September 2, 1947, while at work as a bookkeeper she experienced a full feeling in the lower right quadrant followed by a sharp pain after which she became faint. Upon reclining for a short while she felt better and resumed her work for about thirty minutes. Intermittent fainting ensued accompanied with a bloody vaginal discharge necessitating her return home where she resorted to sedatives and enemas for three days. However, because of increasing distention, dysuria and pain, her physician was summoned; he immediately sent her to the hospital.

Physical examination revealed a well developed, white female weighing 126 pounds. Her blood pressure was 100/68, pulse 130, temperature 99°F. and respiration 26. The abdomen was quite distended and tense; the umbilicus was rather prominent with a circumscribed area of bluish discoloration (Cullen's sign). Increased rigidity over both lower quadrants was present with dullness over flanks. Vaginal examination disclosed a bloody discharge. On movement of the cervix severe pain was experienced. A doughy, crepitant mass was palpated in the pouch of Douglas which was continous with bulging in the right adnexal region; no mass was found in the left adnexal region.

Laboratory findings were as follows: red blood cells, 3,000,000; hemoglobin (8.5 Gm.), 50.4 per cent; color index, .81; 10,000; lymphocytes, 20; monocytes, 2: neutrophiles, 76 and eosinophiles, 2. The urine revealed no unusual findings; the Kahn test was negative.

A diagnosis of ruptured right tubal pregnancy was charted and an immediate abdominal section was performed.

Spinal anesthesia and sodium pentothal were used. On incising the peritoneum a gush of bright red blood ensued. A large mass of clots filled the pelvis and on delivery of the right fallopian tube a rupture in the distal third was found with attached blood clot and tissue. Slight active bleeding was present. The left oviduct was found to be engorged, with a dilated distal portion containing an embryo surrounded by a lobular brown mass which ruptured as a result of operative manipulation.

Bilateral salpingectomy was instituted followed by a transfusion of 500 cc. of blood; autotransfusion was not thought advisable.

The pathologic report (supplied by Dr. E. W. Townsend) was as follows: The specimens consisted of both fallopian tubes. (Fig. 1.) The left tubo-ovarian mass, located at the distal end of the fallopian tube, was dilated and measured 40 mm. in diameter. It was open on one surface and presented a reddish brown color. The open surface had a lobular appearance. In the center of this mass and attached to it was a fetus

measuring 10 mm. in length. The oviduct varied in diameter from 3 to 10 mm. Blocks for microscopic sections were taken just proximal to the mass described, the distal portion of the ruptured fallopian tube with fetus being preserved as a gross specimen.

The microscopic sections showed a hemorrhagic infiltration of the wall of the fallopian tube associated with the presence of masses of fibrin and blood cells within which there were degenerative changes and deposition of hemosiderin.

The second specimen consisted of the right tubal mass and of a clot of blood and tissue measuring 65 mm. in its greatest diameter. The tubal mass measured 130 mm. in length and varied from 8 to 30 mm. in diameter. In the distal third of the oviduct there was a rupture and an expanded portion which measured 35 mm. in diameter. No fetus was grossly identified in this specimen of the right tube and accompanying tissue.

The microscopic sections of the right tubal tissue and blood clot showed numerous cross-sections of chorionic villi and masses of syncytial cells mingled with portions of the fimbriated end of the fallopian tube in which hypertrophic changes, congestion and edema were notable.

The diagnosis was bilateral tubal pregnancy.

The patient was discharged on the twelfth postoperative day.

SUMMARY

A bilateral tubal pregnancy is reported with rupture and intraperitoneal hemorrhage from one oviduct coexistant with an unruptured pregnancy in the opposite oviduct.

As to superfecundation or superfetation in relation to the reported case, this we pass on to the more speculative.

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REACTIONS FOLLOWING INJECTION OF VARICOSE VEINS

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THE proper use of sclerosing agents unquestionably has value in the treatment of varicose veins. It must be remembered that the sclerosing agent destroys the endothelial lining of the vein initiating the formation of an adherent clot. It causes in effect a localized, controlled chemical thrombophlebitis which is accompanied by a variable degree of increased vascular irritability and spasm.

Successful sclerosis depends upon proper selection of cases, proper technic and adequate postoperative care. These factors have been discussed in a previous communication.[1] If a vein is to be permanently obliterated, the amount of clot must be held to a minimum and the vein kept collapsed until healing has taken place. This requires prolonged wearing of elastic bandages or stockings and is true whether ordinary office injections or operative retrograde sclerosis are contemplated. I believe that many unsatisfactory results have been due to neglect of this disagreeable phase of the treatment.

It is observed that with proper selection of cases most of the abnormal reactions to sclerosing agents can be avoided. Thus no serious reactions have occurred in the past two years in the course of treating a considerable number of patients.

Allergic Reactions. Some people are sensitive to the ordinary sclerosing solutions. These include sodium morrhuate, sodium ricinoleate and ethylaminoleate. Those patients exhibiting other allergies such as hay fever, asthma and hives should always be suspected and sensitivity tests made.

CASE REPORTS

CASE I. A. K., age forty-seven was admitted to the New York Post-Graduate Hospital, J94896. The diagnosis was bilateral varicose veins and varicose ulcer of the left ankle. The patient suffered from hay fever and asthma. She was sensitive to ordinary adhesive tape and tests showed allergy to sodium morrhuate and sodium ricinoleate in test doses of 0.2 cc. intravenously. Bilateral saphenous ligation was performed on July 21, 1945, without retrograde sclerosis. Subsequent injection with quinine and urethane was successful. The ulcer has remained closed to date.

Comment. A probable catastrophe was avoided by preliminary sensitivity tests. Patients who have had injection therapy in the past sometimes acquire sensitivity. In these cases a test dose of 0.2 cc. of the sclerosing solution is injected before treatment is begun.

CASE II. A. M., age forty-nine, was admitted to the Midtown Hospital, D-881. The diagnosis was bilateral varicose veins, duration twenty-eight years, with edema and ulceration of right leg.

A left saphenous ligation was performed on September 19, 1944, and retrograde sclerosis with 3.0 cc. sodium morrhuate was carried out. On September 20, 1944, a right saphenous ligation was done; retrograde sclerosis with 4.0 cc. sodium morrhuate was done. She had an uneventful postoperative course. Her maximum temperature was 100.8°F. Five weeks later two residual varices were injected each with 1.0 cc. sodium morrhuate. Two hours later generalized urticaria and mild asthma developed. Subsequently the patient was tested with small doses of sodium ricinoleate and of ethylamineoleate and similar sclerosing solutions but milder reactions took place. No further sclerosis was attempted.

Comment. This acquired sensitivity must be anticipated when a long period is allowed to elapse between injections. In my experience this is the earliest appearance of an acquired sensitivity. I have never had any difficulty with injections

made three to four weeks following retrograde sclerosis.

Reactions Due to Pyrogens. Retrograde sclerosis is performed with a ureteral catheter. If any blood is allowed to remain within, it may cause a foreign protein reaction. In addition broken down blood is an excellent culture medium for bacteria. A case has come to my attention in which a sharp chill and a transient Bacillus coli bacteremia followed retrograde sclerosis. Cultures taken from washings of the catheter grew B. coli.

Reactions Due to Improper Dosage. At the present time relatively small doses of sclerosing agent are used with the retrograde technic. I never use more than 5 cc. on one side and never sclerose more than one side at a time. If too much is injected, the solution puddles and may actually erode the vessel wall causing a sterile abscess. Excessive dosage may also result in pigmented spots or streaks to a greater degree than necessary. It also causes unnecessary pain and fever.

There is a great variation in the reactivity of these patients to their medication which can only be determined by observing the test reaction. It is better to inject an inadequate dose than to bring about an uncomfortable or alarming situation by injecting too much.

Reactions in the Presence of Vascular Disease. Patients with clinical vascular disease should have no sclerosis under any circumstance. Most of these patients have highly irritable vascular systems and sclerosis increases the vasospastic element. In this group I would include patients with thromobophlebitis, Raynaud's disease, Buerger's disease and all those giving symptoms of vascular irritability such as cold, clammy extremities with painful, abnormal response to cold. Sclerosis should never be attempted in the presence of obliterative arterial disease. The added spasm may reduce the caliber of the involved vessels or their collateral bed to a critical degree resulting in gangrene.

Case III. Y. B., age thirty-five was admitted to the New York Post-Gradutae Hospital, J86875. The diagnosis was right femoral thrombophlebitis with ulceration of ankle and bilateral varicose veins. For eight years the patient had had recurrent attacks of phlebitis in her right leg, the first attack occurring during pregnancy. Ulceration on the medial aspect of the right ankle had been present for one year. Bilateral varicose veins were present and were more advanced on the right side. The patient was admitted July 5, 1943, for preliminary elevation and soaks. Sedimentation rate was 35 mm. per hour.

A left saphenous ligation was performed on July 12, 1943, with retrograde sclerosis. A catheter was introduced 35 cm. and 12.0 cc. sodium ricinoleate were injected as the catheter was withdrawn. The next day her temperature rose abruptly to 104.4°F. The left leg became hot and the sclerosed vessels tender. There was no gross edema. Hot packs were applied and the temperature returned to normal in ten days. On July 28, 1943, a right saphenous ligation without sclerosis was performed. The course was uneventful. Subsequently she developed signs of vasospasm and one year later two bilateral paravertebral novocain nerve blocks were performed with considerable benefit.

Comment. No sclerosis should have been done in this case. It undoubtedly caused the alarming temperature response besides aggravating later vasospastic phenomena, nor would we now use as much sclerosing solution under any circumstance. Another case was observed in which a saphenous ligation with retrograde sclerosis had been performed two years following an acute femoral thrombophlebitis. Here no marked reaction occurred but for six months there was increased edema, pain and skin moisture all indicative of vasospasm.

Case IV. E. T. age thirty-seven, was admitted to the Midtown Hospital, C-10648. The diagnosis was bilateral varicose veins and recurrent superficial phlebitis of right leg. For eight years this patient had had episodes, recurring once or twice yearly, of swelling, redness and pain of the right leg. The onset of these attacks was sudden and accompanied by

chills and fever. They yielded promptly to conservative therapy including bed rest, hot packs and sulfonamides. In between attacks the legs swelled on prolonged standing and she noticed progressive enlargement of the veins. Sedimentation rate was 8 mm. per hour.

On June 5, 1944, a left saphenous ligation was done. Retrograde sclerosis was accomplished with 4.0 cc. sodium morrhuate. Two days later she experienced a sudden chill and her temperature rose to 104.2°F. The right leg became swollen, red and painful. Hot packs and sulfadiazine promptly cleared the situation. On June 9, 1944, the patient's temperature became normal. About three weeks later she was readmitted and a right saphenous ligation was done without retrograde sclerosis. Her postoperative course was completely uneventful.

Comment. I believe reactivation of the superficial thrombophlebitis was precipitated by reflex vasospasm induced by sclerosis of veins in the opposite leg.

CASE V. A. E., age forty-six, was admitted to the New York Post-Graduate Hospital, J85700. The diagnosis was bilateral varicose veins and atypical Raynaud's disease of the upper extremities.

On June 20 and 22, 1944, bilateral saphenous ligation with retrograde sclerosis was performed. The postoperative course was characterized by massive sclerosis of both greater saphenous systems, pain and temperature to 103.8°F.

Comment. Although the result as far as the varicosities was concerned was excellent, this patient's Raynaud-like syndrome became worse during the following year. It was undoubtedly aggravated by the use of sclerosing agents. I believe no sclerosis of any sort should be attempted in this type of patient.

CASE VI. H. K., age twenty-nine, was admitted to the New York Post-Graduate Hospital, J88121. The diagnosis was bilateral varicose veins. These veins had been noticed nine years previously following birth of a child. There was no history suggesting phlebitis. For three years there had been considerable pain with edema of the legs on prolonged standing. She was extremely nervous and apprehensive. The extremities were moist and cold.

On October 20, 1944, a left saphenous ligation with retrograde ligation was performed. A catheter was introduced 50 cm. and 3 cc. of sodium ricinoleate were injected. The following day a similar procedure was carried out on the right side. The afternoon following the second operation the patient's temperature rose to 105°F. and the next day rose to 107.4°F. Massive bilateral sclerosis of the entire greater saphenous systems took place. There was no evidence of femoral thrombophlebitis. Conservative measures including the use of papaverine were instituted and six days later her temperature returned to normal. She was discharged on the ninth postoperative day.

Comment. Although the contraindications to retrograde sclerosis in this case were not too clear-cut, it is believed more attention should have been paid to the signs of heightened vascular irritability. Incidentally, an excellent result was obtained.

Homans[2] postulates that some reactions following sclerosis are due to femoral thrombophlebitis. I have not been aware that this is so and believe that immediate and frequent ambulation prevents this serious complication.

SUMMARY

The problem of abnormal reactions following the use of sclerosing agents in the treatment of varicose veins is discussed. Various types of reactions are discussed and illustrative cases are presented.

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HYPERTENSION COMPLICATED BY SPONTANEOUS SUBARACHNOID HEMORRHAGE*

A PLAN OF MANAGEMENT

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IN recent years a significant change has occurred in the method of managing spontaneous subarachnoid hemorrhage. Formerly the emphasis in treatment was entirely on bedrest and supportive measures. After recovered patients had been followed-up for periods of time, it became evident that recurrences of hemorrhage were common and mortality increased with recurrence. Pathologic studies called attention to the existence of ruptured intracranial arterial aneurysm as the lesion very frequently the cause of subarachnoid hemorrhage. The background was thus supplied for the pioneer work of Dandy[1] who then began to treat these patients with a direct surgical approach in the attempt to cure the patient of the aneurysm. Surgery now has a proper place in the management of subarachnoid hemorrhage.

As interest in spontaneous subarachnoid hemorrhage grew, it became apparent that this type of cerebrovascular accident was frequently associated with arterial hypertension. Hirschfeld, Tornay and Yaskin[2] found high blood pressure in 66 per cent of their series of fifty patients. In other reports of collected cases the incidence of hypertension was substantial but not as great.

It was also found that the presence of hypertension increased the mortality from subarachnoid hemorrhage. In Dandy's large series of ruptured intracranial aneurysms there were twenty-five patients with hypertension; all twenty-five died. In a review of the literature Forrest[3] collected 168 cases; in only two instances did persons with systolic blood pressure above 200 mm. survive an episode of spontaneous subarachnoid hemorrhage.

Since hypertension adds to the gravity of the prognosis for these patients, it is reasonable to emphasize this factor in any plan for long range management of such cases. For a number of years we have been impressed with the significant reductions in blood pressure maintained by hypertensive patients who have been treated with the operative procedure of bilateral supradiaphragmatic splanchnicectomy. Among the many cases of essential hypertension in which patients were treated with this operation at the University of Michigan Hospital, there were seven patients who had previously sustained a definite subarachnoid hemorrhage. The subsequent course of these seven patients forms the basis for this report which recommends the use of splanchnicectomy in a plan of treatment for hypertensive persons who sustain a subarachnoid hemorrhage.

SURGICAL ISOLATION OF INTRACRANIAL ANEURYSMS

Subarachnoid hemorrhage of non-traumatic origin may be the result of several conditions, namely, a ruptured intracranial

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† Deceased

arterial aneurysm, extension from a massive intracerebral hemorrhage, meningeal inflammation, bleeding from a neoplasm or a blood dyscrasia. But a ruptured intracranial aneurysm is by far the most common cause. In the large series of Richardson and Hyland[4] careful autopsies performed in fatal cases revealed ruptured arterial aneurysms present in 90 per cent; they thus believed they were justified in assuming that practically all their cases of spontaneous subarachnoid hemorrhage were due to aneurysm, including those patients who survived. Sahs and Keil[5] also have stated that intracranial aneurysms account for most of these cases.

Thus in most instances of spontaneous subarachnoid hemorrhage the cause is a pathologic lesion which can be isolated from the cerebral circulation by appropriate surgical methods. However, the existence of an aneurysm and its location should be clearly defined before a craniotomy is performed. Localizing clinical signs may occur in subarachnoid hemorrhage but the presence of localizing signs is no assurance that an arterial aneurysm will be found nor does it fix the exact site of the aneurysm if one is present.

Intracranial Angiography. Although there is a fixed clinical syndrome characterizing most aneurysms, precise localization and diagnostic confirmation of the aneurysm are obtained only by intracranial angiography. This procedure has been used extensively at the University of Michigan Hospital since 1941 to enable a precise diagnosis of vascular lesions.[6–8] The carotid arteriogram performed with 25 per cent thorotrast is utilized routinely on all patients in whom a ruptured intracranial aneurysm is suspected as soon as the acute phase of the clinical picture has subsided. It has been employed without danger to the patient; neither morbidity nor mortality attributable to the procedure has occurred in this hospital.

Recurrence of Hemorrhage. The absolute need for a surgical method of curing the patient of his intracranial aneurysm is apparent from consideration of the significant recurrence rate of subarachnoid hemorrhage and of the high mortality of recurrences. In Hamby's[9] series there were thirty-two patients admitted in a secondary attack of bleeding; their mortality was 72 per cent. A second hemorrhage occurred in fifty of the 150 patients in Magee's[10] series; it should be pointed out that ninety-eight survived the primary episode. Fifty recurrences in ninety-eight possibilities constitutes a very high recurrence rate. There was a 64 per cent mortality among the fifty recurrences.

In the series of Wolf, Goodell and Wolff[11] there were forty-six patients; 52 per cent had recurrent attacks. The mortality in the twenty-four patients who had recurrent subarachnoid hemorrhages was 41.8 per cent. Wolf and associates combined their cases with those of Magee and found that of the seventy-four patients who had recurrences, forty-one happened in the second to fourth week; of the forty-two patients who died of recurrence, twenty-eight died during this period. They thus conclude "if patients with subarachnoid hemorrhage could be treated effectively and safely by surgical procedures in the first two weeks after the hemorrhage, the threat of recurrent hemorrhage and death in approximately 70 per cent of all patients with subarachnoid hemorrhage could be removed."

SPLANCHNICECTOMY PERFORMED SUBSEQUENT TO SUBARACHNOID HEMORRHAGE

Bilateral supradiaphragmatic splanchnicectomy has been performed on seven patients with essential hypertension in each of whom a spontaneous subarachnoid hemorrhage had occurred from two months to three years before sympathectomy. Each case has been followed up for long periods of time. The clinical data of these cases are summarized in Table 1. Each patient presented the characteristic clinical picture of subarachnoid hemorrhage, further confirmed by the spinal fluid findings. The

TABLE I

CLINICAL DATA IN SEVEN CASES OF ESSENTIAL HYPERTENSION COMPLICATED BY SPONTANEOUS SUBARACHNOID HEMORRHAGE

Case	Age at Time of Hemorrhage	Sex	Symptoms	Neurologic Signs	Blood Pressure at Time of Hemorrhage	Spinal Fluid	Blood Pressure prior to Splanchnicectomy	Result				
								Living or Dead	Survival since Hemorrhage (yr.)	Survival since Splanchnicectomy (yr.)	Recent Blood Pressure	Remarks
I J. W.	42	M	Severe headache of sudden onset; convulsions; projectile vomiting	Papilledema; nuchal rigidity; nystagmus; diminished reflexes	185/130	Xanthochromic pressure 160 mm.	150/120	L	14	13	152/98	No recurrence of bleeding; lives a normal life as a dairy farmer
II H. S.	36	F	Sudden onset of severe headache; blindness; delirium	Stiff neck; stupor	230/140	Bloody pressure 370 mm.	192/120	D	10	9	.....	Her blood pressure averaged 165/95 for 9 yr. until she sustained a cerebrovascular accident and died
III C. P.	46	M	Sudden onset of severe occipital headache; nausea; vomiting; right hemiparesis	Nuchal rigidity; positive Kernig	240/150	Bloody pressure 420 mm.	236/164	L	8	8	190/125	No recurrence; lives an active life
IV W. B.	37	M	Severe headache; diplopia; vertigo; nausea; vomiting	Papilledema; extraocular palsy; nystagmus; ataxia; nuchal rigidity; positive Kernig	240/130	Xanthochromic pressure 350 mm.	210/130	L	8	8	151/104	No recurrence; works hard
V I. B.	38	F	Sudden excruciating headache; nausea; vomiting; thick speech	Nuchal rigidity	185/115	Bloody pressure 220 mm.	195/118	L	10	7	184/112	No recurrence; lives a normal life
VI F. R.	48	M	Sudden onset of severe headache; nausea; vomiting; generalized convulsion	Disoriented and confused; extraocular palsy; absent knee and arm jerks; nuchal rigidity; positive Kernig bilaterally	250/160	Bloody pressure 400 mm.	210/130	D	7	4	.....	No change following operation; died of cerebrovascular accident
VII M. B.	36	M	Sudden onset of severe occipital headache; nausea; vomiting	Coma; nuchal rigidity; internal and external ophthalmoplegia right eye; positive Kernig	160/102	Bloody pressure 450 mm.	178/116	L	3	2	136/88	No recurrence; works as a truck driver

episode of bleeding was treated in this hospital in three instances prior to the time that arteriography was included in our program of management. In three cases the initial hemorrhage was treated in other hospitals. A carotid arteriogram and subsequent craniotomy were performed in Case VII.

Case VII. M. B., age thirty-six, was admitted on May 9, 1945, complaining of severe headache and stiff neck. Three weeks previously he had noted sudden onset of severe headache and stiff neck. The following morning he noted that his right eyelid drooped and his neck was stiff. Headache, nausea and vomiting persisted at bedrest and two days prior to admission his wife found him comatose.

Physical examination on admission revealed a blood pressure of 160/102 and temperature of 99.2°F. orally. He was restless, confused and made many purposeless movements. There was complete internal and external ophthalmoplegia of the right eye. Nuchal rigidity was marked and Kernig's sign was positive bilaterally.

Lumbar puncture revealed bloody spinal fluid under pressure of 450 mm. of water; the red blood cell count of the fluid was 2,350,000 per cu. mm. With bedrest and mild sedation the patient gradually improved.

A right internal carotid arteriogram obtained on June 2, 1945, clearly defined an aneurysm arising from the proximal portion of the middle cerebral artery. (Figs. 1 and 2.)

Craniotomy was performed on June 4, 1945, by Dr. Carl F. List. The aneurysm, the size of a large pea, was found to arise from the beginning of the right middle cerebral artery. An anomalous, collateral blood supply to the right hemisphere was seen to come off the circle of Willis; this permitted ligation of the middle cerebral artery, thus isolating the aneurysm from the cerebral circulation. Postoperative convalescence was uneventful; no paresis developed, indicating that the anomalous, collateral blood supply to the right hemisphere was adequate.

During the following year there was progressive improvement of the ocular palsy. However, the patient's blood pressure persisted at elevated levels and averaged 178/116. He complained of frequent mild headaches and dizziness. The electrocardiogram was within normal limits and the teleoroentgenogram revealed

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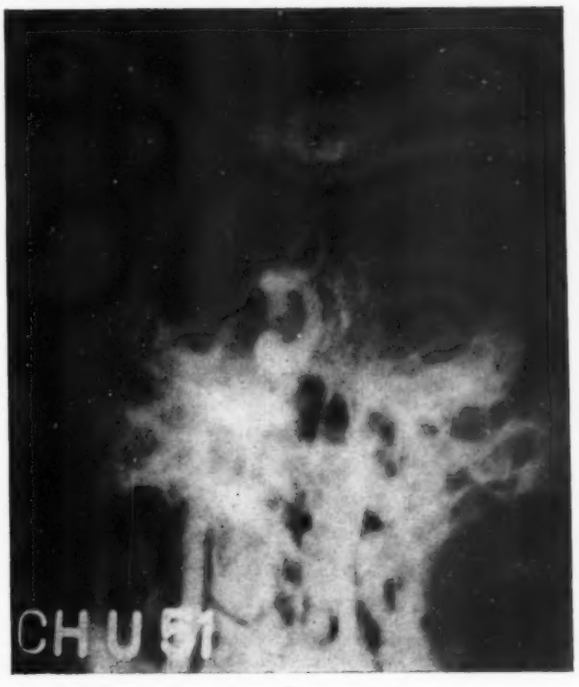

2

FIG. 1 and 2. Right internal carotid arteriograms demonstrating an aneurysm arising from proximal portion of the right middle cerebral artery in Case VII.

normal heart size. Intravenous pyelogram and urinalysis were negative, blood non-protein nitrogen was 37 mg. per cent, urea clearance values averaged 60 per cent and maximum specific gravity on an eighteen-hour concentration test was 1.021. Bilateral supradiaphragmatic splanchnicectomy and lower dorsal ganglionectomy (dorsal 9–12) were done May 9, 1946. The patient was discharged on the ninth postoperative day.

In September, 1948, two years after splanchnicectomy, he was completely asymptomatic. He was maintaining normal blood pressure

levels averaging 136/88 and was earning a livelihood as a truckdriver.

In each instance splanchnicectomy was performed both for the treatment of the patient's hypertensive disease in general and in the attempt to produce a lasting, significant reduction in blood pressure in order to lessen the likelihood of a recurrence of "blowout" of an intracranial aneurysm. High blood pressure undoubtedly was an important factor in the ultimate rupture in each case. Since the ages of these persons ranged from thirty-six to forty-eight years, it is logical to assume that hemorrhage resulted from rupture of a congenital aneurysm in each instance rather than from rupture of an arteriosclerotic vessel. The high mortality resulting from the association of hypertension and subarachnoid hemorrhage warrants an effort to correct the hypertensive state in those few patients fortunate enough to survive the primary hemorrhage. In Dandy's series there were four hypertensives who sustained recurrence of ruptured congenital aneurysms; all four died.

Five of our patients are still living and have had no recurrence, from two to thirteen years after the splanchnicectomy and from three to fourteen years after the subarachnoid hemorrhage. Four have maintained significant reductions of more than 20 mm. of diastolic pressure; the hypertensive levels of the fifth living patient have been uninfluenced by the sympathectomy. The patient in Case 1 has had frequent normal blood pressure determinations during the thirteen years since operation. Another patient, Case 11, maintained a significant reduction of 25 mm. of diastolic pressure for nine years after splanchnicectomy and worked as a stenographer during this time. She then sustained a cerebrovascular accident and died three days later. Both deaths in this group occurred at home and autopsies were not obtained.

A PLAN OF MANAGEMENT

During the acute phase the patient is strictly confined to bed with his head elevated. Therapy is entirely supportive. Sedation is utilized for restlessness and excitement, and analgesics for relief of headache. Fluid requirements are minimal; fluids should be given in amounts no greater than is necessary for adequate kidney function. Lumbar puncture is performed for diagnosis only, with 1 to 2 cc. of spinal fluid slowly withdrawn. Only on rare occasions is lumbar puncture used therapeutically in order to reduce dangerously increased intracranial pressure; spinal fluid is removed very slowly, drop by drop, until approximately half of the initial pressure is established.

After the acute phase has subsided, usually seven to fourteen days, or if there is evidence of persistent bleeding at bedrest, carotid arteriography is performed. If the arteriogram reveals an aneurysm, the ensuing surgical approach will depend upon the location of the aneurysm: (1) Aneurysms of the intracranial portion of the internal carotid artery readily accessible to surgical isolation are those which are located sufficiently below the bifurcation of the internal carotid into the anterior and mid-cerebral arteries to permit trapping of the aneurysm between a ligature of this artery in the neck and another one placed just below the bifurcation. (2) Aneurysms arising from the anterior cerebral artery between the intracranial carotid bifurcation and the anterior communicating artery are isolated by clips placed proximal and distal to the origin of the aneurysm. (3) Aneurysms of the anterior communicating artery often are not amenable to surgical isolation for frequently both anterior cerebral arteries must be ligated in order to isolate such an aneurysm; this latter type of ligation usually results in massive softening of both frontal lobes. (4) Aneurysms of the mid-cerebral artery are attacked directly in the hope of preserving the major circulation to the ipsolateral hemisphere. Naturally, no attempt is made to isolate aneurysms of this artery from the cerebral circulation by ligation. Instead, strengthening of the aneurysmal wall is

sought through muscle packs or other hemostatic tampons. The various operative technics are fully elaborated in a publication by Bassett, List and Peet.[12]

Only by means of arteriographic studies can the indications for the foregoing surgical approaches be determined prior to craniotomy. If the arteriogram reveals no aneurysm, the patient should be confined to bedrest for at least four weeks after bleeding has ceased.

If the hypertensive patient presents himself for the first time following recovery from an episode of hemorrhage: (1) In the presence of persistent, localizing clinical signs and symptoms carotid arteriograms must be taken; and if an aneurysm is revealed, surgical treatment should proceed as outlined. (2) In the absence of localizing signs and symptoms arteriography may be considered optional. However, splanchnicectomy is now recommended in the attempt to produce a persistent, significant reduction in blood pressure.

There is always the possibility that multiple aneurysms may be present; and since one of these may be 'blown out" as the result of persistent hypertension, the patient's essential hypertension should be treated with splanchnicectomy.

SUMMARY

In consideration of the evidence that recurrences of spontaneous subarachnoid hemorrhage are frequent, that mortality increases markedly with recurrence and that mortality is particularly great among those with an associated hypertension, a plan of management directed toward preventing death from recurrent episodes of hemorrhage in survivors of the primary attack has been presented.

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THE YALE SCHOOL OF MEDICINE

THE initial organization of the School of Medicine was completed in 1812 following the passage of a bill by the Connecticut General Assembly in 1810 which granted a charter for "The Medical Institution of Yale College," to be conducted under the joint supervision of the College and the Connecticut State Medical Society. This institution was formally opened in 1813 and the first degrees were conferred the following year. In 1884, with the approval of the Medical Society, the original charter was amended to place the school definitely in the control of the College as the Medical School of Yale College. The name Yale College was changed to Yale University in 1887 and the name of the Medical School was automatically changed. The present name of the school was adopted in 1918.

The godfather of this medical school, whose graduates are found in every state and in many foreign lands, was Benjamin Silliman, sponsor of scientific education in America. He taught the first course in chemistry ever given to candidates for the M.D. degree. Dr. C. N. Hugh Long, one of the nation's leading physiological chemists, has assumed the deanship of the school.

A new Professorship of Oncology is now held by Dr. Samuel C. Harvey, surgeon, and brings together Yale's extensive research on the problems of cancer. This chair of Oncology, one of the first in America, has been made possible by grants from the U.S. Public Health Service and from Mr. Robert Hunter of California. Yale's School of Medicine has been a center for the investigation of cancer problems for many years. At present more than two-thirds of the staff are attacking the cause and possible cure of cancer.

Yale's expanding interest in the field of post-graduate medical education is illustrated by a post-graduate seminar in psychiatry now being held in cooperation with state mental hospital staffs and the Connecticut State Medical Society.

In the same spirit of broader contacts between Yale and the community of which it is a part an Institute of Occupational Medicine has been developed as well as a series of courses in hospital administration which are sponsored by the Yale Department of Public Health in cooperation with the Grace-New Haven Community Hospital.

Yale's affiliations with other hospitals in Connecticut are being expanded in accordance with the general plan for increasing training opportunities and providing a broader pattern of service.

The Yale School of Medicine has long been known for the importance it places on its investigative work which remains one of the keystones of its educational policy.

The total enrollment for 1947–1948 in the Yale School of Medicine is 380. Graduates from 175 institutions are represented in this number. Students are enrolled from forty-five different states and from fourteen foreign countries.

The Yale School of Medicine; entrance, Institute of Human Relations. (Yale University News Bureau.)

New Instrument

CLOSED ANASTOMOSIS BY THE YOUNG CLAMP AFTER COLON RESECTION

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DURING the past two decades there has been marked progress in surgery of the large bowel. The management of carcinoma in the several parts of the colon has been thoroughly reviewed by MacFee,[1] with a comprehensive bibliography. We wish to report a group of cases in which closed aseptic anastomosis has been used after resection of colon carcinomas and discuss certain aspects of the technic.

Progress has been due to many factors such as the applications of physiology, chemotherapy and safer anesthesia which are common to all surgery. However, special technics and instruments have played some part. The resection rate is over 80 per cent and is steadily rising. The hospital mortality of those resected should approximate 5 per cent and the percentage of five-year clinical arrests averages 50 per cent or more.

The instruments that were used for closed anastomosis played an important part in the development of safer colon surgery. We believe that the instrument we are using is important now for other reasons than those which were originally ascribed to it. In 1932 one of the authors[2] published details of an anastomosis clamp which is very simple and which has been in constant use ever since. (Figs. 1 to 3.) Even if we are to eliminate all possibility of infection by chemotherapy, we believe that this clamp still adds very much to the ease and safety with which an anastomosis either between large and small, or between two ends of large or of small, or even an end-to-side anastomosis can be done. When properly used it allows the surgeon and the assistant to hold the bowel with no immediate contact by hand to keep the approximation accurate and to allow the suture material to be used with all possible fineness. It prevents a diaphragm formation. This has been proved by a careful follow-up of our cases. In a few cases in which the postmortem examination after operation allowed the area to be examined carefully, there was no diaphragm formation such as had been described and such as had made trouble in other forms of anastomosis. The fact that the bowel is held firmly by the clamps is very important because the stitches cannot be pulled too tightly. Where the two ends of the bowel are held by a basting thread suture, that anastomotic suture can easily be so tight that it will cut through the tissue when the inevitable postoperative swelling and edema adds more tension. Also, there is no basting thread to break and be left behind as has been reported altogether too often. In all of the cases in which we have used this type of clamp or in which there has been any report that has come to our knowledge, there has never been either a diaphragm formation or a leak or any difficulty which could be laid to the instrument as such. We consider it as a very important part of the armamentarium in the use of intestinal anastomosis of any type.

It seemed desirable to make a careful check of the results we have obtained by applying this method of resection with

closed anastomosis. Our records of the last 100 consecutive private patients with neoplasms of the large bowel or rectum were examined. Of these 100, all patients were hospitalized; ninety-eight of the patients were hospitalized at the Faulkner Hospital. The question of operability was first investigated. It was found that ninety-six patients had undergone ninety-nine operations. Four patients were refused surgery because of advanced age and advanced degrees of growth.

The question of resectability was next investigated. Resection had been considered justifiable if there was a reasonable chance of removing grossly apparent carcinoma. Any reasonable surgical procedure should be attempted which offers the patient with obstructing carcinoma some hope of cure or more comfortable living. It was found that seventy-six resections of the primary growth had been performed on seventy-four patients. This percentage should be a little higher, we believe. Some of the earlier rectal carcinoma cases in which a colostomy was performed would now have an attempted palliative resection done.

Of the resected cases, forty-five patients had resections with anastomosis and restoration of bowel continuity. It is with these forty-five patients that this study is primarily concerned. The remaining patients underwent resections resulting in permanent colostomies, most of which were one-stage Miles' resections.

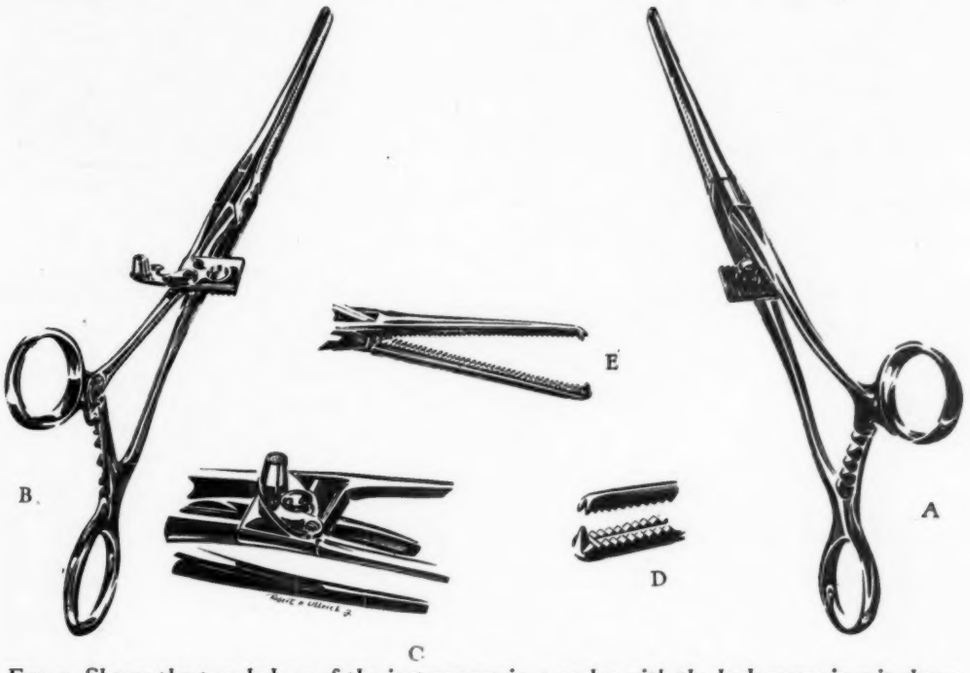

FIG. 1. Shows the two halves of the instrument in A and B with the lock open; in C it shows the simple method of locking the two together uniting the ends of the bowel in close approximation. The details of the jaws are shown in D and E because the toothed end is necessary to prevent the bowel slipping at the edge where the clamp is put in close relation to it. The central groove allows one to be sure that the clamps hold and allow the cautery to sever the bowel in an aseptic fashion close to the clamp.

RIGHT COLON

Right colectomy has been performed in two stages in fifteen patients with carcinoma of either the right colon or hepatic flexure area. There was no hospital death in this group. Thirteen patients are living and well. Six have had an opportunity to survive more than five years and all six are apparently free of disease. The clamp method of anastomosis as described in the illustrations was used in thirteen cases. In one case, because of perforation of the growth into the pericolic fat with a good-

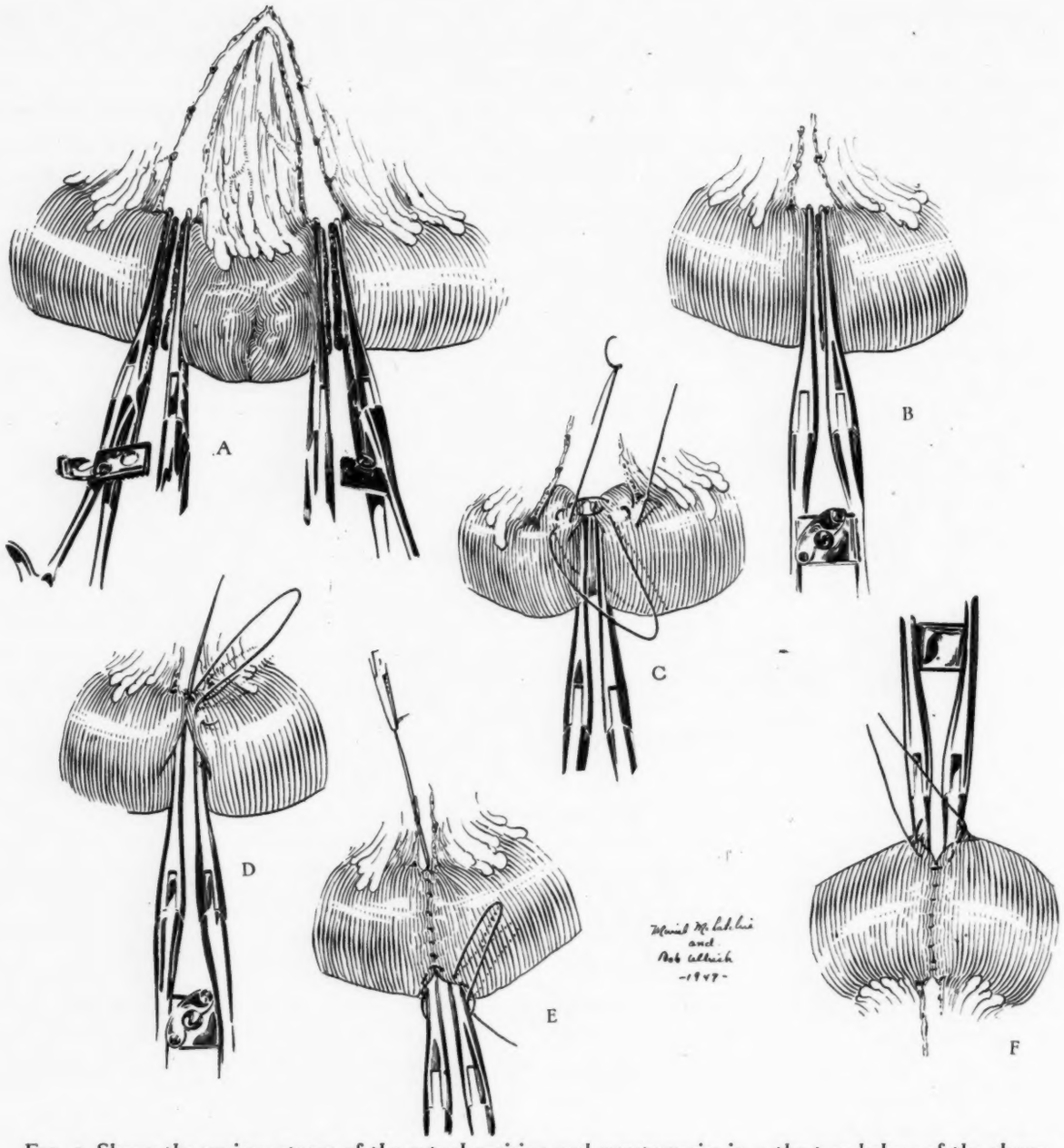

FIG. 2. Shows the various stages of the actual excision and anastomosis; in A the two halves of the clamp should have the tip with the serous surface of the bowel extending just beyond it but the other clamp in any position that would completely close the bowel; in B it shows the tumor removed and the two halves of the clamp locked. Again it should be emphasized that the extreme tip of the clamp should have the serous surface just barely beyond it to facilitate locking all that serous surface over the clamp. A guide stitch is then placed as in C pulling the serous surface over the tip of the clamp. A running stitch is then started on the distal side of the guide stitch and running up each side of the bowel and caught at the top. In D and E the two ends of the two sutures are tied and the tension held by the assistant as the clamps are loosened (still locked together) and withdrawn. This leaves a tight suture which merely needs to be reinforced by a few interrupted mattress sutures.

sized abscess, a Mikulicz exteriorization was done. In one case, that of an extremely tiny, elderly woman, an open anastomosis was done. In one case, done prior to these fifteen, a one-stage palliative resection in the presence of a malignant fistula following rupture of a carcinoma of the cecum with abscess was attempted. This patient died of sepsis. This represents the only mortality in the right colon group. Only two patients have died of disease. We repeat that the remaining thirteen are living, well and free of disease and that there are six who have had an opportunity to survive more than five years. Of the extensive resections which were done in this group, two are worthy of comment. One man had infiltration and involvement on the serous surface of his small bowel and had a resection of 3 feet of his small bowel with a right colectomy. This man gained 40 pounds and had very good palliation but after a year slipped downhill and died of liver metastasis. He is one of the two patients now dead; his treatment might well be classed as a palliative resection. Another patient in whom there was extension through the bowel wall into the ureter and retroperitoneal tissue of a grade IV growth with small pericecal abscesses had an extensive resection involving the parietal abdominal wall and a segment of ureter. This patient is well and free of disease seven years after operation.

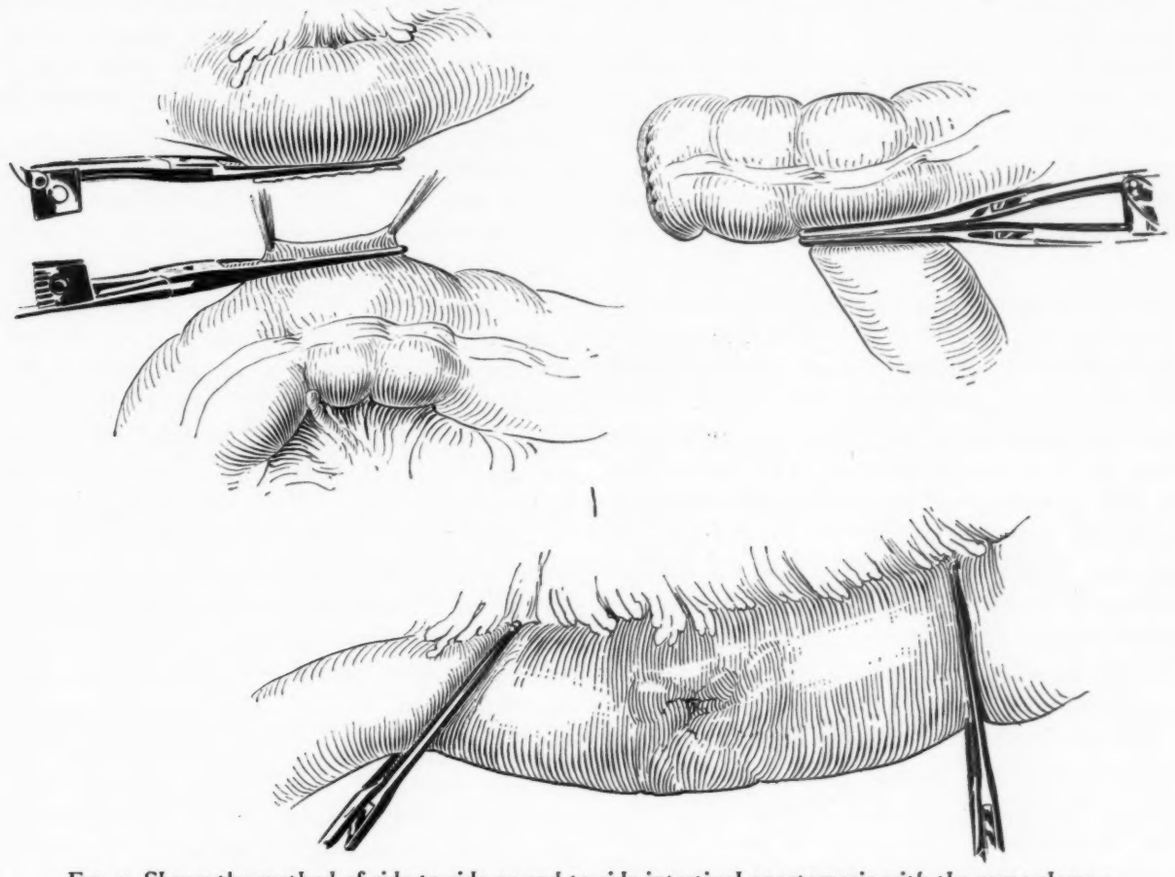

FIG. 3. Shows the method of side-to-side or end-to-side intestinal anastomosis with the same clamps.

LEFT COLON

In this group of cases in which the growth involved the splenic flexure, left or sigmoid colon and upper rectosigmoid there was a total of thirty resections in which a primary end-to-end anastomosis was performed. In this group there were two hospital deaths. The first death occurred very suddenly in the case of a

seventy-five year old man after the abdominal incision had been made prior to starting the resection of his growth at the splenic flexure. However, some traction had been exerted on the bowel in which the growth was located, which might have contributed to his sudden demise; aside from this it was considered an anesthetic death. This case was accepted by the medical examiner and there was no hospital autopsy. The other death occurred in the case of a seventy-nine year old man with a large carcinoma of the transverse colon. This patient remained in very poor general condition after his cecostomy. Resection was postponed for thirty-eight days. At the time of resection considerable plastic peritonitis was found. This patient died one day postoperatively. At autopsy there was no evidence of any leak at the suture line. In four cases resection was performed in the presence of small hepatic nodules believed on palpation to be metastatic lesions. These patients all died of malignancy in one and a half to fifteen months after their resections; their treatment should be separatedly grouped as palliative resections.

Of seventeen patients who have had an opportunity to survive more than five years, there are five now living and well, free of disease. Eight patients have died of malignancy, four are untraced for the full five-year period but three of these were known to be well for shorter periods than five years. In regard to those patients who have survived for a shorter time than five years, five are now living and well, apparently free of disease, one has died of cancer and another of causes other than cancer.

Anastomosis was done by the closed clamp technic in twenty-nine of the thirty resections. In three cases anastomoses were made below the pelvic floor between the upper rectal ampulla and the sigmoid; it was in one of these that the open method was used.

These figures are presented merely in evidence that there had been no delayed stricture formation and that a reasonably wide excision was made by the clamp technic as evidenced by the adequate number of patients cured of their disease. In summary, the closed clamp method as described was used in forty-two anastomoses after resection for colon carcinoma. The hospital deaths are outlined briefly. The end results seem adequate; no strictures or leaks have occurred. The clinical arrests are above the average in the right colon group and slightly below the commonly reported average in the left colon group. We believe that the better results in the carcinomas of the right colon may be attributed to the better removal of regional lymph nodes possible on this side. Attention should be paid to a more radical lymph node removal when resecting carcinomas from the descending colon and sigmoid colon.

CONCLUSION

Closed anastomosis with the Young clamp by the method described has proven safe and advantageous when restoration of continuity can be performed after resection of carcinoma of the colon.

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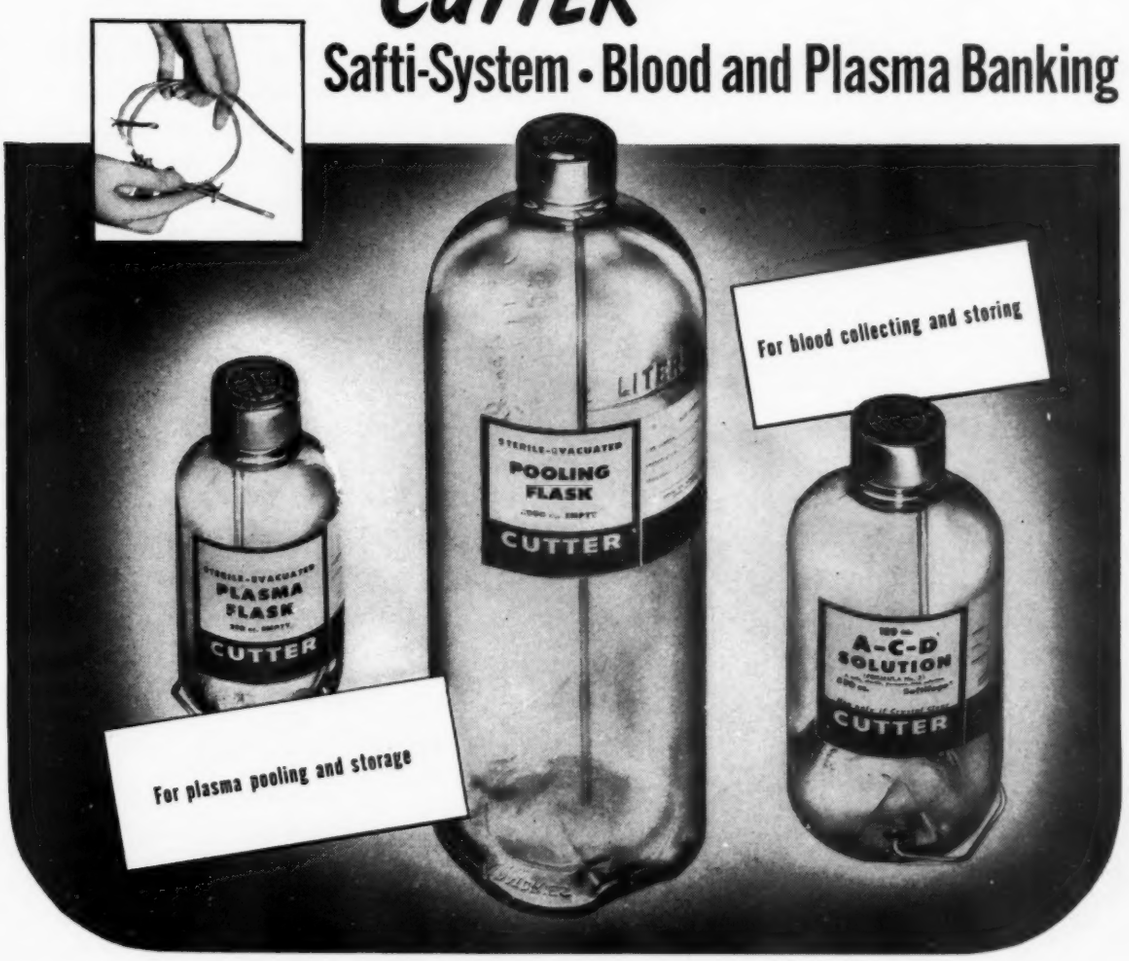

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*New and Nonofficial Remedies, Philadelphia, J. B. Lippincott, 1949, p. 234.

PARKE, DAVIS & COMPANY

MEDICAMENTA VERA

Outside the Ivory Tower

(*continued from page 16*)

their public mouthpiece, the Hearst Press, picked out the medical scientist as their 'defamation goat,' they picked out a person who didn't have the time, the temperament or the weapons to fight back.

"But," Dr. Carlson added, "events of recent years, ranging from actual violence, slowing of research and stoppage of medical training to general and personal villification on its lowest level have forced the doctors and scientists to take time, to adjust their temperaments and to utilize the democratic weapons of organization and law to fight back. Now the fight on the legal front is just beginning and this issue is joined. Two million dollar libel suits have been filed by medical scientists against the Hearst Publishing Company and William Randolph Hearst. Two more are pending and other suits will follow if necessary. This is the beginning of a policy which took shape when a nationwide legal committee was formed by the National Society for Medical Research to advance medical research and to protect the medical worker and his institution against what appears to be an organized program to villify and slander medical science and scientists."

JAMES T. CASE, Professor of Radiology at Northwestern University Medical School, wrote the Foreword of "Bone and Joint Radiology," a new work by Emerik Markovits, M.D. It is published by The Macmillan Company. We can do no better than quote in part from this Foreword: "Every roentgenologic report logically consists of two sections, the first part dealing with an accurate description of the aberrations from the normal as seen on the screen or in roentgenograms, the other part consisting of an interpretation of these findings. If several experienced radiologists each dictated a separate report on the same case, the factual descriptions of the roentgenograms should scarcely vary; but the interpretation of these findings may differ considerably, depending on the experience of the individual physician-interpreter.

"The present work ("Bone and Joint Radiology") is a very commendable effort to marshall in outline form the various radiologic findings in diseases and injuries of bones and joints thus facilitating the analyses of these findings in any given case and improving greatly the prospect of their correct interpretation in terms of clinical medicine. One has to turn through the pages of this most helpful tabular exposition of roentgen findings to realize the potential value of this orderly array of roentgenologic contributions to diagnosis. In further pursuing the methodical presentation of the x-ray findings there have been provided numerous differential diagnostic tables and classifications which should prove valuable." This is a beautifully done book with 446 pages, 616 illustrations and it is indexed. We believe it should be in the office of every physician doing x-ray work. Also, the young man in practice, as well as interns and residents, would do well to possess a copy and give it serious and repeated attention.

IF we were asked to recommend a good work on obstetrics and gynecology, general surgery, proctology or on the heart and peripheral vascular disease, we would suggest a selection of several volumes. If we were asked to recommend a book on the intervening aorta, we would choose Dr. Nathaniel E. Reich's "Diseases of the Aorta" which is published by The Macmillan Company and costs $7.50. As far as we know it is the first comprehensive text which deals with the problems connected with the cardiovascular apparatus. It is an excellent work. After a Foreword by William Dock, M.D., and an Introduction, the book covers the following: The Aorta, Congenital Anomalies Involving the Aorta, Atherosclerosis of the Aorta, Syphilis of the Aorta, Diseases at Origin of the Aortic Lumen, Dissecting Aneurysms, Occlusions of the Aorta, Rare Diseases of the Aorta, Diagnostic Procedures (not requiring x-ray equipment), Diagnostic Procedures (requiring x-ray equipment), Antibiotics, Anticoagulants and an Index. There are ample and up-to-the-minute references at the conclusion of each chapter. The book is illustrated. It contains the newer surgical procedures. To medical students, particularly interns and residents, and to the established man in the practice of his profession we urge that this work be carefully read. It is sound science, shows painstaking work and deserves to be named one of the outstanding works of the year.

(*continued on page 30*)

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FOR INFORMATION ADDRESS

MEDICAL EXECUTIVE OFFICER, 345 West 50th Street, NEW YORK 19

Outside the Ivory Tower

(*continued from page 28*)

THE Buffalo Surgical Society announces the award of the Roswell Park Medal to Dr. Evarts A. Graham for outstanding surgical achievement.

ALL working surgeons, especially surgical residents and young surgeons still on the lower rungs of the ladder, could study with profit the pages of J. Markowitz's "Experimental Surgery, Including Surgical Physiology" (second edition). This book fills an important need for laboratories using animals for medical research purposes. One will read a description of nearly all of the common operative procedures used in the experimental laboratory. It will have a wide appeal among veterinary surgeons and is a work that should be on hand in all surgical centers, research surgical departments and teaching institutions. The book contains 546 pages, 330 illustrations, a bibliography and an index. The Williams & Wilkins Company publish this book and the price is $7.00.

IN no medical field has there been such outstanding advances as in the field of vascular surgery. Thus we welcome an authoritative work on this subject by a trained worker in that field, Dr. Gerald H. Pratt's "Surgical Management of Vascular Diseases." There is a Foreword by W. Wayne Babcock, M.D., Temple University, Philadelphia, with whom at one time Dr. Pratt was associated. In this work treatment is emphasized but essential anatomic, diagnostic and pathologic facts are given thorough consideration. The illustrations, two of which are in color, are excellent and many of them were made right in the operating room. A bibliography is at the end of each chapter and there is a workable index. We highly recommend this book to surgeons, internists, hospital house-men and undergraduate students. Lea and Febiger put out this book for $10.

BOTH the surgeon and the general practitioner will profit greatly by consulting or reading throughout Dr. W. N. Kemp's looseleaf (115 pages) practical work, "The Value of Hormones in General Practice," published by Burges Publishing Company. In this day and age when hormones are prescribed willy nilly for every human ailment from flat feet to dysmenorrhea, it is refreshing to peruse a common sense treatise, backed by scientific facts, on this subject. Surgeons will find value in the part that deals with hormonal-chirugical treatment of cryptorchidism.

THOSE interested in autobiography and who knew or know of the late Dr. Meyer will want a copy of the "Autobiography of Dr. Robert Meyer (1864–1947), A Short Abstract of a Long Life, with a Memoir of Dr. Meyer" by Emil Novak, M.D. It is enough to tell you it is delightful reading and a modest recital of a full and absorbing career. It was published by Henry Schuman and the cost is $2.50.

IF you like to read medical history during an odd pause or before retiring for the night, we recommend J. D. H. Widdess' (Librarian and Lecturer in Biology, R.C.S.I.) with a Foreword by William Doolin's F.R.C.S.I. (Hon. Librarian, Royal College of Surgeons in Ireland), "An Account of the Schools of Surgery —Royal College of Surgeons, Dublin." It is a well written book (117 pages, illustrated and indexed). You will not find your time wasted. The Williams and Wilkins Company put out this book for $5.

UNLESS you know all there is to know about hernia and if you are an intern or surgical resident, you will find much meat in Dr. Alfred H. Iason's "Synopsis of Hernia." It is a compact work of 500 pages (well illustrated, indexed) covering the subject in full. It is highly recommended to those interested in this subject. The cost is $6.50 and Grune and Stratton are the publishers.

WE now turn to three historical works. Charles C. Thomas, Publisher, has done H. Winnet Orr's "On the Contributions of Hugh Owen Thomas of Liverpool, John Ridlon, M.D., of New York and Chicago, and Sir Robert Jones of Liverpool and London to Modern Orthopedic Surgery." There is a Supplement on Ridlon and His Share in Moulding Orthopedic Surgery by Arthur Steindler, M.D. This book is well written and interesting, especially to orthopedic surgeons, although anyone interested in medical history and biography will find it good reading; the price, $4.50.

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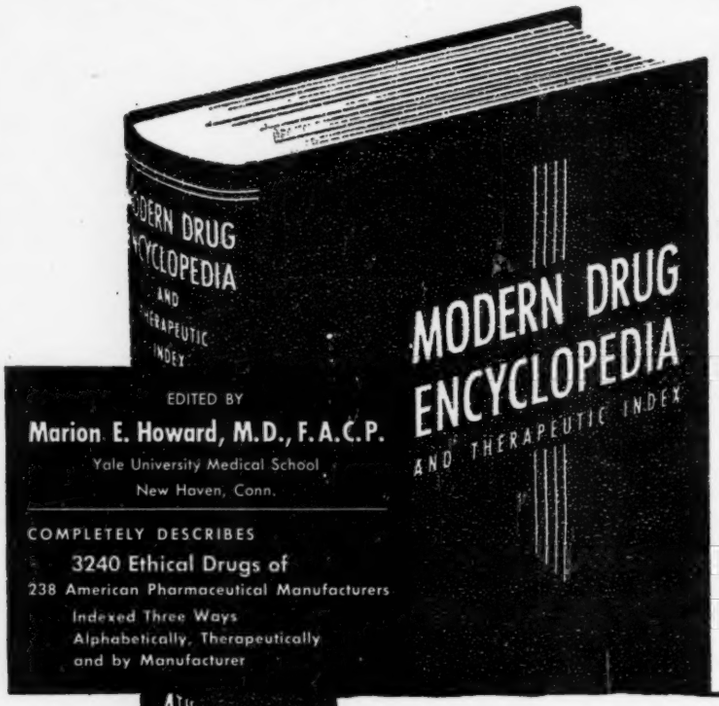

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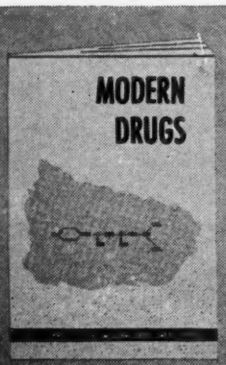

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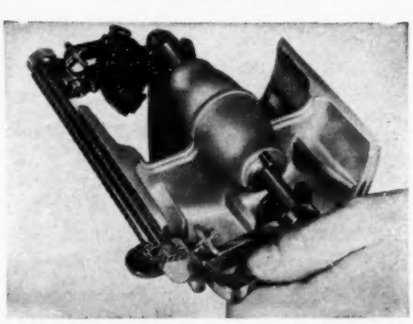

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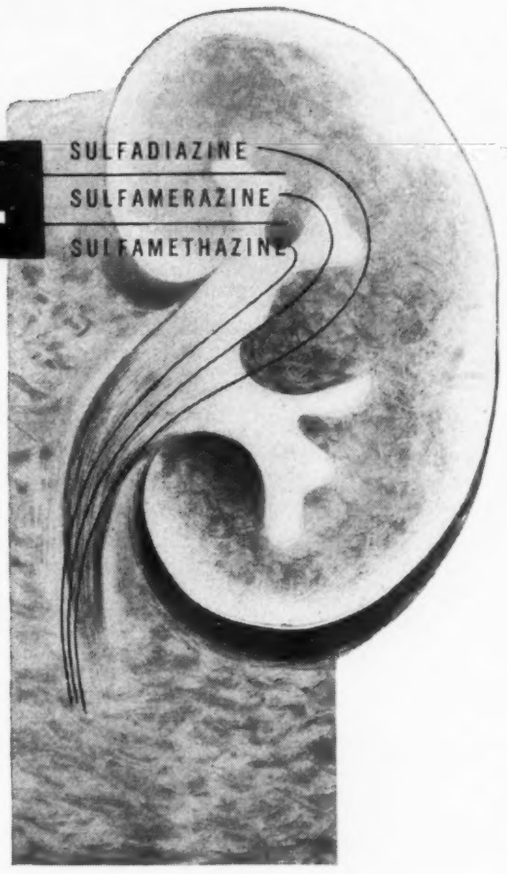

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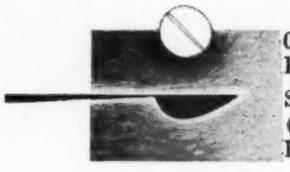

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